SOME OBSERVATIONS ON THE COURSE OF ARTERIES

A Study of Arterial Patterns in Man, as exemplified in the Placenta, the Pulmonary and Bronchial Arteries, the Arteries of the Intestine and of Skeletal Muscle, demonstrating the fundamental relationship between the course of arteries and the function of the organs they supply.

T H E S I S B Y

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INTRODUCTION

Kugel (1928) writes "The blood supply to a tissue is often a key to the understanding of pathological processes which may occur in the tissue".

Owing to this reason, perhaps, and owing to the dramatic results of its injuries and diseases, we find that the cardio-vascular system has been very extensively and exhaustively studied for centuries.

In the dissecting rooms, one is struck by the very frequent variations seen in the course of the arteries, so frequent in fact that Ruysch (quoted by Segall {1923}; and Graham and Cannell {1933}) propounded a dictum "No given artery distributes itself in exactly the same manner in any two or more cases". And Sir John Fraser, in his review of malignant disease of the large intestine (1938) when describing the colic arteries, writes "Statistics suggest that what the anatomist would describe as an abnormality is almost as frequent as the disposition which anatomy claims to/

to be normal". Textbooks of Anatomy give descriptions of the commoner variations; almost every number of current anatomical journals describe examples of unusual vascular arrangements; and any demonstrator of Anatomy, during a term's work, can collect a list of unusual arteries from the cadavers in his own dissecting room.

Flint (1923) for example described accessory hepatic and cystic arteries; Patten (1927) variations of the coeliac axis. To quote observations in the Anatomy Department of Edinburgh University, in one case, the left profunda femoris artery arose just below the inguinal ligament, passed between a bifid origin of the saphenous nerve, and continued on chiefly as the medial femoral circumflex artery; in another, the left accessory meningeal artery split the inferior dental nerve, the auriculo-temporal nerve having one root in this subject. But quotations on this subject could be endless; while some anomalies are apparently unique, such as the anterior tibial artery passing forward lateral to the neck of the fibula, described by Velpeau in 1839, others are largely anatomical commonplaces.

On/

On viewing these anomalies, a whole host of questions naturally occur to the enquirer. At what sites are aberrant vessels commonest, in the viscera, limbs, etc? In which divisions of the system, in the heart, arteries, veins or capillaries? If there is one aberrant vessel, is there a tendency to numerous such vessels in the same subject, either bilaterally or unilaterally? Are anomalous courses of vessels associated with intrinsic vascular anomalies, such as bifid origins, cirsoid aneurysms, congenital aneurysms, tortuosities, kinks, loops? Are anomalous vessels associated with anomalies of the organs they supply?

Some of these questions, of course, and similar ones, have been answered. Thus Senior, in his paper entitled "An Interpretation of the recorded arterial anomalies of the human leg and foot" (1919) notes "the presence in the adult limb of vessels which from their course and termination were clearly recognizable as persisting embryonic arteries" and also states "It is questionable whether even the most local deviation from the course of normal arterial development manifests itself entirely within the limits of a single adult artery..... in consequence of the very intimate/

intimate interdependence of the alternate arterial routes which successively become available during the progress of development, it happens that arterial anomalies affect general arterial regions rather than individual vessels".

Anomalous vessels, then, may be modifications of normal developmental processes, but what has caused these modifications? "Some do not represent any known condition met with, either temporarily or permanently, in man or in other animals. Their occurrence cannot at present be adequately explained, and their retention in the adult is entirely dependant upon their utility". (Cunningham 1943). Let us note that last word in particular "utility" .

With Ruysch's dictum in mind, and considering the frequency of anomalous courses of vessels, and the various questions listed above, one is liable to think that vascular distribution is a haphazard affair, and the normal course of the arteries is not governed by any rule.

Further investigations, however, and a narrower scrutiny, make one arrive at a very different conclusion. John Hilton, "hot-foot in the/

the application of the doctrine of utilitarianism to the structure of the human body" as Sir Arthur Keith (1919) described him, stressed, in his classic book, 'Rest and Pain' (1863) "the great precision which marks the supply of arterial blood to some parts of the body". He considered the definite distribution of arteries a point not sufficiently dwelt upon, and amplified his statement with the description of some examples in detail: "The branches distributed from the subclavian trunk apart from its continuation to the upper extremity, are distributed with one single purpose, viz, to supply all the parts concerned directly and indirectly in the process of respiration..... that is the simple object of the distribution of the subclavian" Again, "The essential purpose of the internal maxillary artery is to build up or nourish those parts directly necessary or accessory to the process of mastication, so that it may be fairly called the masticatory artery".

Hilton's hypothesis has been supported by other anatomists. Shellshear (1921) states that "the association between blood vascular distribution and function is no mere coincidence/

coincidence..... We are justified in using the blood supply as a confirmatory evidence in investigation of function..... nuclei or areas of cortex which serve a separate function have their own arterial supply" Shellshear gives us two rules "(a) Arteries are laid down with a definite relation to function, and (b) The distribution of arteries obeys some ontogenetic or phylogenetic law".
He also refers to John Hunter's observation of the angles at which branches of the cerebral arteries arise specially so in order to retard or allow a freer motion of the blood in them. Keith (1933) Franklin (1932 & 1938) Wood Jones (1917) on the Anatomy of Veins, Stopford (1916, 1930) on the Functional Significance of the Arrangement of the Cerebral Vessels, and Louis Gross (1918) in his work on the Kidney, can also be quoted in support of Hilton.

With these authors' researches as a guide, it will be evident then that there are very definite rules and restrictions in force regulating the branching of the arterial tree, and that there is a very good reason for the alternative arrangements that obtain.

The vascular system must be a more or less "mobile" system, otherwise the exigencies of/

of inflammation would evoke no salutary response and the alterations required for healing purposes would be impossible. Vessels form readily in adhesions, rapidly in tumours (Rutherford Morison 1896); the success of grafting operations, for example omental grafts (Wilkie 1911 and Bothe 1929) omentopexy (Morison and Drummond 1896) and cardio-omentopexy (O'Shaughnessy 1937) depends on the formation of vascular channels.

The vascular system is a purely subservient system, liable to be varied by the needs of the organs it supplies, as contrasted with the commanding system, the nervous system, that most stable, conservative of systems, as Cunningham (1899) has described it in his study of the significance of anatomical variations.

If then we examine an arrangement of vessels, whether it be loops, circles and arcades of arteries (Woollard and Weddell 1934); the angles at which arteries leave their parent trunk (Hunter) or at which veins join larger venous channels (Stopford 1930) etc., we must consider that there is a definite reason underlying that particular pattern. This pattern may guide us to discover the function of the particular organ supplied, and lead us to suppose that similarly supplied organs may have some function/

function or mechanism of action in common. The comparison of the blood supply of a well known sphincter such as the pylorus, with another more debatable one such as that at the lower end of the bile duct, might help us to decide, by the similarity or otherwise, whether it is a true sphincter we are dealing with, as Oddi (1887-8) and Archibald (1919) suppose, or whether it is purely a valve as Coffey describes it (1928). And this comparison might be applied to other regions, i.e. the ureteric vesical orifices.

This close association of form and function holds good throughout the system, from the largest vessels to the capillaries. This has been well stressed by many authors. As Mall (1905) says, "It is the organ itself which determines the quantity, the rate of flow, and the pressure of the blood flowing through it", and to quote Gross again, "The importance of the circulatory arrangement in an organ in relation to its construction and function is evident and well recognized. Knowledge of it furnishes to a great extent the key to knowledge of function. This is nowhere better illustrated than in the Kidney".

The arterio-venous channels (Krogh 1929, Hoyer 1877, Grosser 1902, Heimberger 1925) the capillary/

capillary content of organs, the disparity in size of the afferent and efferent glomerular arterioles (Schafer 1938) the thinness of the walls of Intracranial arteries (Maximow and Bloom 1938) all indicate very definite specific relation to function. Thoma (1896) has pointed out the well defined laws of histo-mechanical principals that influence the development of the vascular system, and the numerous intensive researches in recent years on the blood supply of the heart, all stress the intimate association of organs and their vascularization. "Blood vessels grow into the myocardium when the latter has need for more blood" state Beck and Tichy (1935). And Schlesinger (1938) writes "Anastomoses always develop readily whenever and wherever arteriosclerotic narrowing or occlusion causes obstruction in the coronary artery circulation..... such zones of anastomotic circulation are not distributed indiscriminately but in each individual heart the anastomoses were specifically designed to compensate for the occlusion."

We can amplify Hilton's idea by further observations, which help to confirm and justify/

justify the belief in a precise vascular plan.

(1) Vessels lying loose in beds exposed to constant movement are tortuous and so can stretch with the surrounding parts without undue strain which might narrow the lumen and impede the blood flow. Examples of this are the facial, the superficial temporal and the occipital arteries. Another example is the testicular, long and straight on the posterior abdominal wall; but as soon as it reaches the place where it is liable to be moved about, in the scrotum, it becomes tortuous. The vessels in the limbs, where movement is going on constantly, might be expected to be equally tortuous; but they are straight, and so are applied close to the bone, covered by fibrous sheathes.

(2) Vessels going to organs liable to fairly rapid gross changes in form, are also tortuous, to allow for stretching; for example, the uterine. The terminal vessels to the intestines pass in at right angles to the longitudinal course of the gut, not parallel with it, and so are not subject to a concertina-like squeeze every time a peristaltic wave passes down. "The arteries of a muscle branch freely, and between the branches there are very numerous/

numerous anastomoses forming a primary network..... also a secondary network from the threads of which the arterioles branch off, generally at right angles, to the muscle fibres, and at very regular intervals.... ... The whole of the vascular system is beautifully adapted to the changes of muscle contraction; the arterial and venous network insure supply and drainage of almost every point, even if a number of anastomoses are temporarily blocked" (Krogh 1930, quoting Spalteholz 1888).

(3) Arteries run truly to their appointed destinations, and do not waste themselves by giving off odd branches to neighbouring structures, or at any rate, only very rarely; these are generally trifling in character (e.g. the small branches to the subperitoneal tissues in the case of the testicular arteries (Quain, 1899)).

(4) The course of arteries are carefully protected channels, especially when contrasted with the nerves. No artery of any magnitude lies practically bare against exposed bone, as does the ulnar or common peroneal nerve. The hypoglossal nerve courses over the hyoglossus, but the corresponding artery, the lingual, lies deep. And if an artery does lie near a bone, or other rigid structure, liable to crushing or compression by muscle/

muscle action, it is carefully protected; witness the definite foramina in the fascia lata for the terminal twigs of the circumflex and perforating arteries; the hiatus tendineus for the popliteal, the strong tendon of scalenus anterior in front of the subclavian, etc.

Arteries at first sight superficial, on further investigation are found to be better protected than one would suppose. The radial artery at the wrist lies snugly in the concave volar aspect of the radius, and soon disappears under cover of the thumb extensors. The facial artery lies deep to the level of the firm masseter muscle as it passes over the lower border of the jaw; and the muscle first encounters the finger placed horizontally along that border. The accessory nerve passes straight across the posterior triangle of the neck in an exposed position, but the arteries of that triangle are carefully tucked away under muscle edges or bone, the transverse cervical under trapezius, the suprascapular hidden by the clavicle.

One may even state that arteries only come to the surface, or lie unduly exposed when the blood supply to the part they are going to has been adequately insured by collateral vessels; witness the vessels in the scalp, and the free anastomoses of the relatively superficial superior thyroid

arteries/

Fig A.

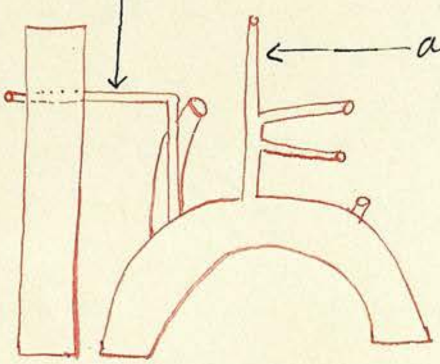
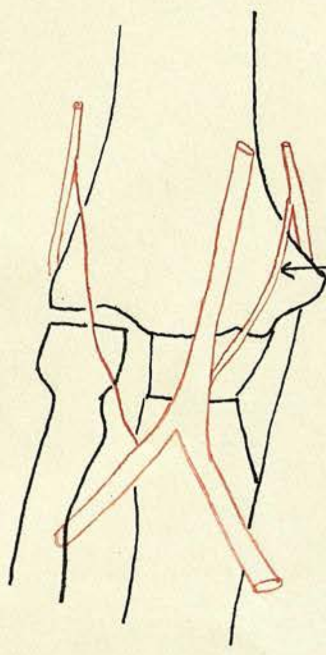

Inferior thyroid artery coming off main subclavian.
ascending cervical artery

Fig B.


anterior ulnar recurrent artery coming off main brachial artery before it bifurcates.

arteries. Every main trunk, approaching the surface, is protected by an awning of fibrous tissue; the clavipectoral fascia over the axillary, the bicipital aponeurosis over the brachial, the fascia lata and femoral sheath over the femoral artery. Where this is less marked, the nerve is accounted less worthy of protection than the artery; for example, the medial popliteal nerve passing superficial to the popliteal artery.

Certain definite rules appear to guide even the minor variations in arterial courses. If in any part of the body there is an arterial arcade or circle, (mesenteric arcade, circulus arteriosus of Willis) then the exact course of the main feeding trunks to this arcade is apt to vary. But the essential basis of the pattern will be the same, though the finer details of the fabric may alter (for example the "marginal artery" of Hamilton Drummond, 1914a, 1914b and Mondor, quoted by Meillere 1927, and its supplying vessels).

Similarly, at the splitting of an "axis" artery (e.g. the thyroid axis) the subdivisions very often vary in their details; in one case the condition in Fig.A was seen. Another example, at the elbow joint, the outline of the anastomotic pattern is very constant, but the feeders/

feeders vary; in one case, the arrangement seen in Fig.B occurred. In development, where one organ develops as an outgrowth of another, it may take with it not only its regular ration of vessels but may also appropriate one or more extra arteries from the original trunks to the organ from which it sprung. This was noted by Lockwood in his paper on the development of the arteries of the abdomen and their relation to the peritoneum (1885) when, quoting Cruveilhier, he refers to the additional vessels that may supply the liver, and says "Generally speaking, structures receive their blood supply by the nearest route, and exceptions to this rule can usually be accounted for".

The above mentioned sorts of variations are very common and are not real anomalies. Even in the formation of the largest arteries, i.e. the roots of the primitive dorsal and ventral aorta variations occur (Cunningham 1943); but these do not in any way affect their physiological function and the destination is eventually reached though the route may be circuitous. One may refer to J.S. Campbell's paper on the coronary system (1928-29) "There is" he writes "with one exception, nothing constant in the distribution of the atrial blood vessels. This is the/

the ramus ostii cavae superiosis. It is always present; it has no constant origin, but has a constant termination".

The variations, may, of course, be sufficiently embarassing to the surgeon, e.g. the accessory cystic arteries described by Flint (1923) in the operation of cholecystectomy, but may on the contrary, in accidental lesions of important arteries, e.g. the hepatic (Behrend 1920) be of value as Segall (1923) has pointed out. He says "When the necessity arises for ligation of a previously normal hepatic artery without delay, as for example because of traumatic rupture..... then the surgeon must be encouraged by the frequent occurrences of anomalies which enhance the collateral arterial circulation of the liver".

A feature in arterial courses, hitherto not discussed, may now be mentioned, namely the arrangement of vessels as a possible guide to determining particular parts of an organ, and also to determine the position of junctional areas. This was investigated by Monks (1903) in his endeavour to obtain a reliable method for determining exact intestinal localisation; he considered the arrangement of the mesenteric vessels had some features which/

which intimately concerned this subject, and he described the variations in the arcades, as we have already quoted. Rothschild (1929) also refers to localisation of a segment of small intestine from a study of its vascularisation. No definite criteria, however, were laid down by these authors. Reeves (1919) and Wilkie (1911) noted the difference of the blood supply of that part of the duodenum derived from the foregut, and the part derived from the mid-gut; but Yule (1927) in his study of the arterial supply to the duodenum stated "Morphologists cannot use the arterial supply to determine the limits of the fore and mid gut".

In the previous pages, then, one has endeavoured to stress the point that Hilton's observations are fundamentally true when he noted "The precision which marks the course of some arteries". One could go further and say "The course and the structure of the vessels have been laid down with exact precision, and are fundamentally correlated with the functions of the organs they supply". One must mention, however, that Turner (1865) wrote "I may state, as a general result of my injections in these (subpleural mediastinal plexus) and other localities, that though the area or domain appropriated to each artery is as a rule, clearly/

clearly defined, yet that where adjacent areas are in contact, the arteries of one almost invariably inosculate with the arteries of another, and under some conditions actually encroach upon the domain of another". This is altogether a different view to Hilton's.

While the explanation for many special developments in morphology is fairly well understood, (e.g. the orientation of venous valves in relation to body surfaces Edwards, 1936) and the presence in some organs of sinusoids, etc., others are still a mystery though there must be a very good reason for them.

Bearing this in mind, let us look at some arrangement of vessels in more detail.

Two common arrangements of vessels at once arouse the investigators attention; the "end-arteries" and the "Anastomoses".

All arteries terminate in the capillary bed. The capillary bed must receive a regular number of feeders to keep it full, like the streams running into a lake. Here, indeed, is a complete anastomotic region; but the channels are small, and cannot compensate if one of the feeders is suddenly blocked. For, by blocking of one of the feeders, the points of fresh entry become too far apart, so that long before the blood has reached the/

the centre of the area whose feeder has been blocked, its vital properties have been absorbed, and it has become loaded with the products of metabolism.

A given area of tissue with its capillary bed must be supplied by a definite number of feeders; the more active an organ, the more vascular it is. In the heart there is "One capillary for each muscle fibre in the ventricular walls, and papillary muscles, and a less abundant supply in the auricular muscle and the Purkinge system" (Wearn 1928). It is, however, the arrangement of the arterial feeders that concerns us especially; it stands to reason that the more solitary one of these is, and the fewer connections it has with its fellows, the more precarious is the nourishment of the bed it supplies.

To arteries running an apparently solitary course, then, the term "End-artery" has been applied; for example, the arteria centralis retinae, a perfect end artery as Cohnheim described it, and still called so by Duke-Elder (1932). The term "End-artery" originated from the classic work of Cohnheim (1872) on the pathogenesis of Infarction. His work is quoted in all studies on this subject, to which, indeed, little essential has been added, and it will be frequently referred to here.

Extracts/

Extracts of his monograph were translated into English for me by M.W. Griffel in 1938 in connection with my investigation.

Cohnheim showed that in many regions of the body embolism and ligatures of single arteries was rendered "Innocuous by the arterial supply and all its numerous anastomoses..... with at the most, functional disturbance resulting of more or less passing nature" but how, in the lungs, "For regulation of disturbed circulation, single anastomoses are not sufficient but a fairly large amount of them is required". In certain areas end-arteries were situated; in the spleen, kidney, retina, brain and the lungs.

He described the effect of blocking of vessels in these organs, and how in the lungs "end-artery is no definite anatomical criterion, as it is in the other organs mentioned, because in the lung we have to take into account individual differences concerning the capacity of dilatation of small vessels, the energy of circulation, etc." We see straight away, then, that there are factors present influencing our definition of "end-artery". Cohnheim then went on to describe the pathology of infarction, to which I will refer later.

There/

There still do not appear to be any definite rules, or definite criteria drawn up, which can be applied to a vessel under examination to enable one to say "This is an end-artery, or this is not an end-artery".

A search through the recent literature does not reveal any specific definition, though Cobb (1931) gives some alternatives: "If, then, one defines "End-arteries" as those arterial branches which in no way connect, even through the capillary bed, with other arterial branches, there are probably no end-arteries in the body, certainly not in the brain. If the definition is that the branch in question must have no connection with other arterial branches (including arterioles) there probably are no end-arteries except a few in the kidney, spleen, mesentery and retina. My own observations on the brain would lead me to believe that there are none there, even in the basal ganglia. If one considers any artery that does not have obvious anastomoses of branches large enough to be called truly arterial to be an end-artery, one may find many such in the organs enumerated, and a few in the brain. Functionally speaking, however, the capillary and arteriolar anastomoses keep the arterial tree from acting as the sole blood supply of any part".

We/

We find many arteries labelled as "end-arteries" and also many opposing views on individual vessels. We have seen Cobb's views on the arteries of the brain; but other authors differ. Beevor (1909) quotes Heubner (1874) as considering the arteries of the basal region of the cerebral hemispheres to be true end-arteries, in the sense used by Cohnheim. "Cohnheim clearly enunciated the theory that infarcts could occur only where there was no anastomoses between arteries" (Cobb 1931). Beevor also quotes Kolisko (1891) on the branches which come from the trunk of the anterior choroid artery and supply the internal capsule. "In one place Kolisko states that they are end-arteries, but later refers to their capillary anastomoses, rendering it difficult to know if he considered them end-arteries or not..... Duret (1874) showed that there are no anastomoses between different arteries penetrating the basal ganglia, and no anastomoses between the cortical and basal arteries, as they are end-arteries..... I also agree with Duret's view that the cortical arterioles become end-arteries as soon as they enter the cortical substance". The last observation is also confirmed by Temple Fay (1925). But Wolff (1936) states "Thus from a study/

study of the cerebral cortex and basal ganglia, in the lower vertebrates and in man there is no evidence for the existence of end-arteries".

In other regions of the body the same conflicting views are found. Hamilton Drummond (1914a) in referring to the blood supply of the large bowel, writes "The arcades of the sigmoids anastomose freely with each other but the lower sigmoid branch does not form an anastomosis with the superior haemorrhoidal artery in this arcade fashion; the superior haemorrhoidal artery forms no anastomotic arcade, but is, so to speak, a terminal artery". He later (1914b) describes De Dietrich's and Sudeck's ligature above the "critical point" on the inferior mesenteric artery, and stresses the insignificance and variability of the middle haemorrhoidal artery, a fact also brought out by Stewart and Rankin (1933). Moynihan on the contrary has written (1913) "It is almost impossible to deprive the cut ends of the colon (when resection is being done) of an adequate blood supply. Mortification due to anaemia is a myth". Pope and Judd (1929) also refer to the "critical point" (the junction of the last sigmoid and the superior haemorrhoidal artery) of Sudeck (1907) and Hartmann/

Hartmann (1909), but say it is a misnomer, and found "instead of a single middle haemorrhoidal on either side to the rectum, there was always three and sometimes five on either side".

In even sharper contrast we have the statements of H. Eisberg (1925) "The vasa recta of the small intestine are not end arteries" and the results of A.J. Cokkinis's (1930) investigations "The collateral circulation stops with the terminal row of arcades. Beyond this there is absolutely no anastomosis, either between the vasa recta in the mesentery or between the ramifying vessels on the gut wall". He refers also to the work of Litten (1875) quoted by Trotter (1913) in support of his statement.

Wilkie (1911), referring to the fact that "In standard works on anatomy the blood supply of the Duodenum is dismissed in a few words" described the supraduodenal artery to the first portion of the duodenum, and thus confirmed Mayo's classic clinical observation of the "Anaemic Spot" there (1908). The supraduodenal artery therefore is physiologically an end-artery.

Further examples could be given of the opposing opinions expressed by different investigators in this field, and reflection shows that this state of affairs is almost inevitable.

It/

It is in the coronary system, perhaps, that we get a hint as to how to describe and define the end-arteries. For, in this localised field, investigated intensively by many workers, (O'Shaughnessey, Kugel, Campbell, Gross, Wearn, etc.) we see the effects of obstruction of the vessels, of different degrees, and at different speeds of formation. A sudden blocking of a coronary artery will produce a necrosis of the part it supplied, a slow and gradual occlusion on the other hand may occur in a similar vessel, and finally completely obliterate its lumen, but no necrosis will occur (Schlesinger 1938). In this case a collateral circulation has developed, adequate to maintain the needs of the area of myocardium supplied. The obliterated artery, then, has not proved itself an end-artery at all; it was only a potential one, and the circumstances which would have made it one did not occur.

Our judgment of the classification of arteries, then, must be based on their functional distribution. This distribution may only be demonstrated in a negative way by the results of the cessation of that function, as seen in pathological lesions./

lesions. It is significant that the process of infarction, and the conception of end-arteries were first clearly described by Cohnheim, when studying a pathological, not a purely anatomical entity. He showed moreover that the process of infarction and the extent of tissue involved depended upon many more factors than simple arterial blocking. We have already referred to one or two factors affecting the lungs. The time factor is important. "The bronchial arteries, important as they may be for compensation of chronic circulatory disturbances in the lung are ineffective in those acute processes we are dealing with..... they do not interfere with the character of the pulmonary branches as end-arteries". The infection factor, especially in the lung, may greatly alter the picture; the retrograde circulation from the veins may congest the embolised area, obscuring a clear-cut picture of arterial block. Cohnheim also noted the fact "that no infarction takes place if the embolised end-artery is beyond a certain size" because the blood coagulates in the bigger veins draining the obstructed area, and renders impossible any kind of further congestion.

A system of end-arteries is contrary to the principles involved in the building of the body/

body mechanism. The attractive organ-unit theory of Mall (1905), "it appears that each organ is broken up into units which are of equal value from anatomical and physiological standpoints" does not really apply in the human frame, as Mall himself admitted when applying his theory to an individual organ, e.g. the liver: "It soon became evident that the liver lobule was not the simplest of all structural units but was extremely difficult to follow in its development".

Mall's theory would explain the possible presence of a great number of end-arteries, and would certainly constitute a simple arrangement of terminal vessels. This would mean a great many organ units with a precarious blood supply, and as we have seen, arterial channels are among the best protected, and most carefully arranged in the body mechanism. Their topography is a more complex affair.

Even as things actually exist, structures whose blood supply is not so well protected as their neighbours' invariably are placed in a position of danger when they are injured or diseased. The blood supply of tendons is just adequate for their needs, and in inflammation is very/

very apt to be obliterated by the pressure of inflammatory products; though, as Kanavel (1939) says "whether this tendon necrosis is due to the great toxicity of the streptococcus infection or great oedema about the effusion into the sheath shutting off the blood supply, may be a question".

The distal four-fifths of terminal phalanges of the hands, (Kanavel), the first inch of the duodenum (Wilkie 1911) the lesser curvature of the stomach (Reeves 1919) are other sites liable to necrosis if the blood supply is interfered with, or a prolonged course of repair, and great difficulty in healing if diseased.

The term "End-artery" then is not really a strict anatomical definition, but is essentially a physiological term, and can be applied to several arteries that are not usually regarded as end-arteries. Muir (1929) writes "The term must not be used in a strictly anatomical sense to indicate that there is no anastomosis, but only in the sense that, when obstruction occurs, the circulation cannot be satisfactorily restored by collaterals". The lower popliteal can quite well be described as an "end-artery" for its collateral circulation/

circulation is poor (Stopford 1924) and gangrene may result from its occlusion (Thomson, Miles and Wilkie 1931). I observed this myself during the War, 1939-45; in several patients with wounds of the popliteal artery, gangrene of the lower extremity resulted in every case necessitating amputation through the thigh.

If a vessel is occluded very gradually, then, the body has time to make a collateral supply, and practically no artery cannot be fully compensated for. If a vessel does not communicate with vessels capable of carrying on the circulation in the part it supplies, in the event of its <u>sudden</u> obstruction (not merely check the retrograde venous flow) then it is an end-artery. Such arteries are relatively few, and can only be correctly studied in the pathology room, in the process of infarction. Confined to a pure anatomical study of a vessel, all one can say of it is that it may be a <u>potential</u> end-artery.

We have discussed some of the sites for these arteries in the preceding pages; they occur in the heart, lungs, brain, kidney, stomach, duodenum and intestine. We can add those of the spleen/

spleen, whose vascular system has been studied repeatedly, and in which there is still some doubt as to whether an open or closed circulation occurs (Robinson 1926; Macneal 1926; Macneal, Otani and Patterson 1927). We could add even the diaphyseal arteries in growing bone, the phenomenon of disease in the metaphysis being essentially that of infarction (H.A. Harris 1930).

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The method by which the body ensures itself against the risk of local death consequent upon interference of blood supply, is in the system of connecting links in the vascular chain known as Anastomoses. Anastomoses are the channels, therefore, which prevent as far as possible the presence of potential end-arteries. Anastomoses are present in many different forms.

The/

The vascular system largely develops as a series of anastomosing channels, and many of the arteries are formed by the joining up of somatic inter-segmental channels. For example, the internal mammary, the superior and inferior epigastric arteries represent anastomoses between the anterior ends of the ventral branches of the somatic intersegmental arteries (Cunningham 1943). The prelaminar, the post-central and the pre- and post- neural anastomoses presist, the latter two aiding in the formation of the thoracic and lumbar portions of the anterior and posterior spinal arteries respectively (Cunningham).

Anastomosing channels form very readily in development; the most striking example is seen in the rare congenital condition of stenosis or coarctation of the aorta, in which there occurs a great enlargement of the anastomoses from the vessels of the head and neck so as to provide a blood supply for the trunk and lower part of the body (Muir 1929).

When this stenosis, of varying degrees of completeness occurs at or immediately below/

below the insertion of the ductus arteriosus, the most extreme development of anastomoses takes place, "enabling life to be carried on with ease and comfort so long as the myocardium remains equal to the task imposed upon it, and the heart's mechanism is intact" (Abbot 1928), but very often with disabling symptoms (E.J.Rooke 1938) and a tendency to haemorrhage due to the abnormally raised blood pressure in their upper extremities, acting on defective arterial walls (Walker and Livingstone 1938). A case is described by Hamilton and Abbot (1928) and a complete Bibliography has been compiled about this condition by Abbot, with quotations and descriptions of the two hundred cases in the literature up to 1928. Similar development of many abnormal compensating branches occurs with the rare congenital stenosis of the abdominal aorta (Maycock 1937).

The stimulus of a diminishing blood supply in an organ, provokes the formation of anastomoses of compensatory nature, either by widening of old channels, or the development of new ones, following the principles of Thoma's histo-mechanical laws. This kind of anastomosis has been/

been well studied in the coronary arteries by Schlesinger (1938) "In a group of thirty-five hearts taken from patients over 56 years of age, a rich anastomic circulation (is found) only (in) those hearts in which there was occlusion of the coronary arteries. Such zones of anastomic circulation were not distributed indiscriminately but were specifically designed to compensate for the occlusion..... anastomoses in the coronary system develop only when and where there is need for them. Then and there they develop quite easily and readily and usually to a sufficient degree to compensate adequately". No situation could be summed up more clearly.

Many authors confirm this. "In thromboangeitis obliterans the anastomotic channel has developed to the utmost (Woollard and Weddell 1934-35). The process of development of collateral circulation is not entirely straightforward, "for the development of an efficient collateral circulation, depending as it does on an active dilatation of the anastomotic arteries, presupposes a comparatively healthy condition of the artery walls..... if the arteries are the seat of disease they/

they are often unable to dilate sufficiently" (Muir 1929). The cause of the obstruction of the circulation through the main vessel moreover may also act equally well on the collaterals. This is commonly seen in wounds. In fifteen cases with vascular injuries of the limbs, eleven developed gangrene (Trevor 1944) "concomitant damage to soft tissues, causing thrombosis in the surrounding tissue and extending to the collateral circulation no doubt contributed to the production of gangrene". The whole subject of peripheral vessels, and their surgery is in fact at present very much to the foreground (Learmonth 1940) and arteriography has been developed as a procedure for demonstrating in the living subject.

(1) The anatomical arrangement, whether normal or abnormal.

(2) The presence of local irregularities in calibre in arteriosclerosis or Buerger's disease.

(3) Thrombotic blockage of main trunks in obliterative vascular disease.

(4) The extent of the collateral circulation.

(5) The site and extent of arterial aneurysms

and

(6) The site of artero-venous fistulae. (Learmonth 1944 a).

Anastomoses also develop between vessels of organs and their neighbours. The extra-cardiac/

extra-cardiac anastomoses of the coronary vessels, described by Moritz, Hudson and Wearn (1932) and those of the hepatic and the phrenic arteries, described by Segall (1923) and Graham and Cannell (1932) can be given as examples of these.

The possibility of success in grafting depends on the development of similar classes of anastomoses; the success of skin "tube" grafting is further ensured (according to the teaching of J.J.M. Shaw) by compressing one end of the tube to obstruct the inflow, and thus stimulate a copious vascularity at the other end which will form the base when the tube is transposed to its new bed. The growth of vessels in tubed skin flaps, and the establishment of circulation in the flaps has been investigated by German, Finesilver and Davies (1933).

The work of O'Shaughnessey (1933, 1936, 1937), Rutherford Morison (1896), already mentioned, depend on this class of anastomosis. The vitality of tissues which are completely removed from their original position, however, and transposed to a new site, e.g. detached omental grafts, is precarious; for there may not be sufficient/

sufficient time for vessels to grow in before necrosis occurs, the time factor being so important in development of any collateral blood supply. Detached omental grafts are therefore considered almost useless by some, (e.g. Rubin 1911) and others have found that a thin graft, easily vascularized, takes better than a thick one (Finton and Peet 1919). Small omental grafts do take readily, there is no doubt, and are much used in abdominal surgery (C.H. Mayo 1917; Hamilton Bailey 1938). Davis (1917) and Freeman (1916) both found omental grafts took very well, and became well vascularized.

Anastomoses also occur to some degree between splanchnic and somatic systems. The subperitoneal arterial plexus described by Turner "which constitute minute anastomoses between the visceral and parietal branches of the abdominal aorta in the subperitoneal tissue" is an example of these.

Another interesting group is the arterio-venous anastomoses. These have been described as occurring in the human skin (Heimberger, quoted by Krogh 1930) and within the digital bones (Grosser 1902), and are described in Krogh's work on the capillaries. They also occur in the thyroid gland/

gland and G.S. Williamson (1925) writes "It would appear that there is a shunt circuit in the interstitial tissue of the organ". Arterio-venous anastomoses have been extensively studied by Grant and Bland (1929-31) in the human skin and birds' feet, by Moore (1935-36) in the ear of the dog, and Clark and Clark (1934 a,b) in rabbits' ears. These anastomoses are not to be confused with pathological or congenital arterio-venous fistulae, which produce gross deformities (See Harton 1931), or traumatic arterio-venous communications, a subject of great importance due to the War (for an interesting example of this variety see Learmonth 1944 b.). A further example is pointed out by Wearn (1928) in his paper on the role of the Thebesian vessels in the circulation of the heart. "It is clear that three types of connections exist (1) the direct connection between the arteries and the Thebesian vessels (2) the venous connection (coronary) with the Thebesian vessels (3) the capillaries which run directly into the Thebesian vessels..... It is interesting to note that these connections enabled the Thebesian vessels in two cases of old standing closure of the coronary artery orifices to supply the heart muscle with the necessary blood.....

the/

the closure of the coronary orifices in these two cases was a gradual one". Here we have a further example of arteries potentially end-arteries, being completely occluded without untoward results.

Apart from the above well-defined groups, there are different types of anastomoses occuring in the normal adult systemic arterial tree. The reason for the varying patterns of anastomoses is more or less obvious in certain cases, i.e. a mechanism to distribute blood equally and ensure blood supply of vital parts, and in other places to break the force of the full tide of the blood stream.

Reeves, for example, (1919) quoting Waldeyer, considers the plexus of tortuous vessels in the submucosa of the stomach a mechanism to offer a resistance to the blood stream when the organ is contracted and empty, and not requiring a rich supply of blood; and notes, how, when the stomach becomes full, the spiral curves of the arteries are straightened out, and the flow of blood to the mucosa made less difficult, which is exactly what is required for digestion activity.

The reason for some of the finer details in Anastomoses, however, is obscure. Monks, in his attempt at Intestinal Localisation (1903) has noted that opposite the upper part of the bowel, "the/

"the superior mesenteric artery's branches are united by a system of primary loops, but as we proceed down the bowel secondary loops become more numerous, and further down, the secondary loops become still more numerous..... while opposite the lower part of the gut the loops generally lose their characteristic appearance and are represented by a complicated network". The reason for this alteration in pattern awaits explanation.

The circle of Willis, formed by the anastomoses of arterial trunks of relatively large size, represents, as Cohnheim says (1872), "an excellent and even typical arrangement of anastomoses".

The series of varying arcades from which straight arteries arise, is the pattern of anastomosis which obtains in the small intestine.

In the large intestine, feeding arteries run to one long continuous anastomotic channel, the marginal artery of Riolan, Mondor and Hamilton Drummond, which gives off arcades.

Arterial loops, formed by one artery or more, from which spring single vessels, are seen in the kidney, and in the palmar and plantar arteries.

The/

The above are variations of one theme. A second big group is the anastomoses of longitudinal channels seen so constantly in the joint region of the limbs; there may be only one series, as at the elbow (radial recurrent to anterior branch of profunda, etc.) or even three, as at the knee (Stopford 1924-25), i.e. anastomotica magna and descending branch of the lateral femoral circumflex to the superior articular branches of the popliteal; these to the inferior articulars of the popliteal; these to the recurrents of the anterior tibial.

The reason for these arrangements is surely to allow free range of blood across a joint at whatever stage of flexion and extension the joint may be, alternative channels remaining open as their neighbours become compressed.

A third group is the joining of two arteries from opposite sides to form one trunk, "convergent anastomosis", a further precaution to ensure a blood supply to a vital region, seen in the junction of the vertebral arteries to form the basilar./

basilar. This might be taken as an extreme example of anastomoses across the mid line of bilateral symmetrically represented vessels. These anastomoses may be free, as between the branches of the external carotid, or sparse, e.g. between the intrinsic lingual vessels.

We may contrast these with anastomoses between the branches of a single artery. Schlesinger (1938) details examples of these in his study of the coronary arteries, recognising three types of anastomoses (1) Anastomotic channels carrying the blood from one large branch of one of the coronary arteries to another large branch of the same vessel. (2) The arterial channel distal to an obstruction being supplied by blood from the opposite coronary artery. (3) An arterial branch receiving blood from both coronary arteries, a convergant anastomosis.

Such, then, are some of the varieties of anastomoses.

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These are some of the facts regarding arterial courses in general. The account now follows of the investigation of further particular aspects of this subject.

This research fell naturally into several distinct chapters:

(1) The investigation of an end-artery.

(2) The investigation of a debatable area of anastomosis.

(3) The investigation of the arteries of a region where considerable difference of opinion regarding their distribution obtains.

(4) The investigation of the arteries of an organ where the vascular patterns have been less fully described than elsewhere in the body.

The arteries studied in these four chapters were:

(1) The arteries of the placenta.

(2) The pulmonary and bronchial vascular system.

(3) The arteries of the intestine.

(4) The arterial supply of skeletal muscle.

The experimental work for this research was commenced in 1938, when the investigation of the blood supply of the colon and small intestine was begun. Following this, the pulmonary bronchial vessels were studied. Then from August 1939 - October 1945 there was an interruption due to War Service, and the work was not resumed until January 1946, a gap of over six years. The investigation then restarted with the conclusion of the intestinal section, and this was followed by the study of the placenta, and finally the experiments on the blood supply of muscle.

The sections, however, have been arranged according to the anatomical order and not in a chronological sequence.

SECTION I

The Investigation of an End Artery

The vessels of the human placenta were studied as an example, par excellence, of end-arteries. The placental circulation has been investigated by Bacsich and Smout (1938), in 50 placentae. They found that the two umbilical arteries have a constant communication either immediately before or after their entrance into the placenta. No sign of a peripheral anastomosis was obtained, and they do not believe that the two arteries communicate except by the transverse branch; all the arterial branches distal to the transverse communicating branch being end-arteries; "This anatomical fact explains the occurrence of white infarcts..... The transverse communicating branch... plays a most important role in regulating the circulation of this organ. It may have the same functional role as the Circle of Willis to equalise the pressure and establish an equal blood supply to all parts of the brain. The normal and certainly the pregnant uterus carries out rhythmic contractions and it is obvious that during the contraction/

contraction wave the blood pressure in the corresponding part of the intervillous space and cotyledons of the placenta is increased, and the presence of a pressure equalising or "buffer" system is well justified. This arrangement may also be compared with the arterial arcade of the intestines, where the continuous peristaltic movement is comparable to the contractions of the uterus. The transverse communicating branch establishes an equal distribution of blood and regulates the pressure in the placenta, counteracting as a buffer system the effects of uterine contractions".

Technique.

The method for demonstrating the umbilical arteries was the celloidin corrosin injection technique which was employed by Bacsich and Smout. The method has been used frequently before (Narat, Loef, Narat. 1936. Huber. 1906. Hinman, Morison, Lee Brown. 1923) a full description being that of Pettigrew (1934). The injection mass consists of a solution of celloidin in acetone. As acetone readily combines with water, the celloidin is rapidly precipitated out of solution/

solution whenever water is encountered. Thus, by injecting the celloidin solution into the lumen of blood vessels, where moisture is present, one soon obtains a deposition of celloidin, forming a cast. When the parenchyma of an organ with vessels so injected is macerated, the celloidin cast is revealed.

The celloidin cast technique was used in all four sections of this research, and very numerous experiments were performed. It is therefore pertinent at this point to describe in detail the actual modifications employed.

The materials for the injection mass were acetone and celloidin. Camphor is advised to render the cast less friable, but many of the specimens in this research were produced without it. The celloidin used was pure celloidin, either pure celloidin wool (G.T. Gurr, London) or celloidin shreds (Schering, Berlin). Old X-ray films have been used with success, but after several trials they were not employed, as the casts obtained with them were too fragile, crumbling and tending to form loose hollow casts in the interior of the big vessels, rather than firm solid rods.

Varying/

Varying strengths of celloidin can be employed: 3% for finer vessels to show detailed structure, 10% for coarser injections to show main vascular trunks (Pettigrew 1934). The percentage of celloidin employed as a routine was 6% unless otherwise specified in individual experiments. This percentage with pure celloidin, gives an adequate cast of vessels down to the finest arteries perceivable by the naked eye. In some specimens, after the first injection with 6%, a stronger percentage, 12% was used to make a strong cast of the main trunks.

Colouring Agents

Many pigments have been employed as colouring agents with the celloidin cast technique. The resin dye alkanin (Melville & Hunter, Edinburgh) has been found to be eminently satisfactory. Alkanin is readily soluble in acetone, and unaffected by the corrosion agent employed, hydrochloric acid. It gives a strong deep red colour to the celloidin. As a contrasting dye, when two different vessels were being injected in the same organ, trypan blue has been employed. Trypan blue is not soluble in acetone/

acetone though highly soluble in water. It therefore forms a suspension in acetone of a dull grey-blue colour, not a striking contrast to the rich red alkanin at first. Once injected, however, the blue colour becomes more pronounced as the injection mass comes in contact with the moisture in the vessels.

The celloidin alkanin solution is injected by a closed pressure bottle worked by an electric motor system. Prior to injection of the celloidin, the specimen for injection is perfused with tap water slightly acidulated with HCL. This acidulation helps to haemolyse any blood clot (Bacsich and Smout 1938). A glass cannula is inserted into the intended vessel, and this connected with a pressure bottle which in turn is connected to the electric motor. A mercury manometer in the circuit records the pressure in millimetres of mercury. Thorough preliminary perfusion with water is necessary to wash out the blood clot in the arteries, and thus avoid patchy irregular filling. Perfusion is also essential to detect divided vessels, which can therefore be caught by artery forceps and pressure clips and ligated. This preliminary perfusion is especially important when dealing/

dealing with the placenta which is practically full of blood, and requires prolonged washing through before the return outflow from the vein becomes clear.

After perfusion with water, the celloidin alkanin mixture is injected. The whole success of the injection is totally ruined unless a closed circuit can be maintained - hence the importance of ligation of as many divided vessels as possible, detected by the preliminary injection of water. Once the injection of the celloidin is started, divided vessels missed at the preliminary investigation are more easily detected owing to the bright red alkanin dye. They must all be secured, as the smallest leak will produce a deflation in the system and cause a flabby friable cast, crumbling at the gentlest handling. All the rubber tubes and glass connections must also be free of old dried celloidin, otherwise portions of this are apt to be loosened, stick in the system and prevent the full pressure being maintained, and consequently prevent the correct filling of the vessels, and give a totally erroneous picture of their pattern, and a friable cast.

All/

All connections must be absolutely secure before starting pressure bottle injections. Fresh specimens, floated in warm water throughout the injection, give the best results. In water the arteries of the part receive a more equal distribution of the solution than when lying on a hard surface, where the weight of the specimen tends to squeeze the vessels of the lower part and distort the pattern. In warm water also, the celloidin acetone mixture flows more freely and any leak is readily detected.

The pressure at which the celloidin acetone mixture is injected has been varied in different experiments. It has been found most suitable to use a high pressure at first, 450-500 mm mercury, to reach the finer vessels quickly otherwise coagulation occurs before the finest radicles are reached, and an imperfect, untrue appearance is obtained. In some experiments, to ensure this filling of the finer vessels, pure acetone is injected before proceeding with the injection mass, as advocated by Bacsich and Smout (1938). This modification ensures the passage of the celloidin to the extremities of the finer vessels before precipitation; if the vessels are filled with water, precipitation will tend to occur more readily in spite/

spite of a high pressure of injection. Narat, Loef and Narat (1936) achieved the same object by a preliminary insufflation with air.

It will be noted that the pressure employed (450-500 mm Hg.) is considerably higher than the normal arterial pressure in life. This raised pressure is essential to pervade the vessels in the stiff inelastic tissues from the cadaver. The pressure is turned on, the stop cock adjusted to produce this high pressure, and the rubber tube which is to go to the cannula held between finger and thumb till completely filled up to the point of occlusion. Then it is quickly slid into the cannula. If not done in this way, air bubbles will occur in the circuit, which will produce distortions in the cast by inadequate fillings, and weaken it. The high pressure (450-500 mm Hg.) is maintained for a variable period, generally half an hour. The motor is then stopped and the pressure left on in the closed circuit via the cylinder in the circuit, which maintains the pressure by compressed air. If there is any leakage, then the pressure falls quickly and a poor, weak cast is obtained. The preliminary half hour should have completely filled the vessels and the pressure left on should maintain this/

this, without much more celloidin entering them; the pressure in the closed system should fall very gradually. It should be left on at least 12 hours, by which time all the celloidin should have been precipitated out of solution, and formed a solid cast of the vessels.

The specimen, after injection, should be left 12-24 hours for setting. Setting or coagulation has occurred in the finest radicles almost immediately; but proceeds much more slowly in the bigger vessels, on which the stability of the cast depends. The specimen can therefore be left several days if required. If the main vessels do not appear to be properly filled, a further injection of 12% or 15% celloidin can be injected to fill them.

After setting, corrosion is required. Corrosion, or maceration can be obtained by various means. Bacsich and Smout mention

(1) by commercial HCl diluted with 1/6 water

(2) by ordinary macerating methods

(3) by pepsin and HCl digestion

(4) by 1% KOH in an incubator at 56°C.

Pure hydrochloric acid is advocated by Pettigrew,

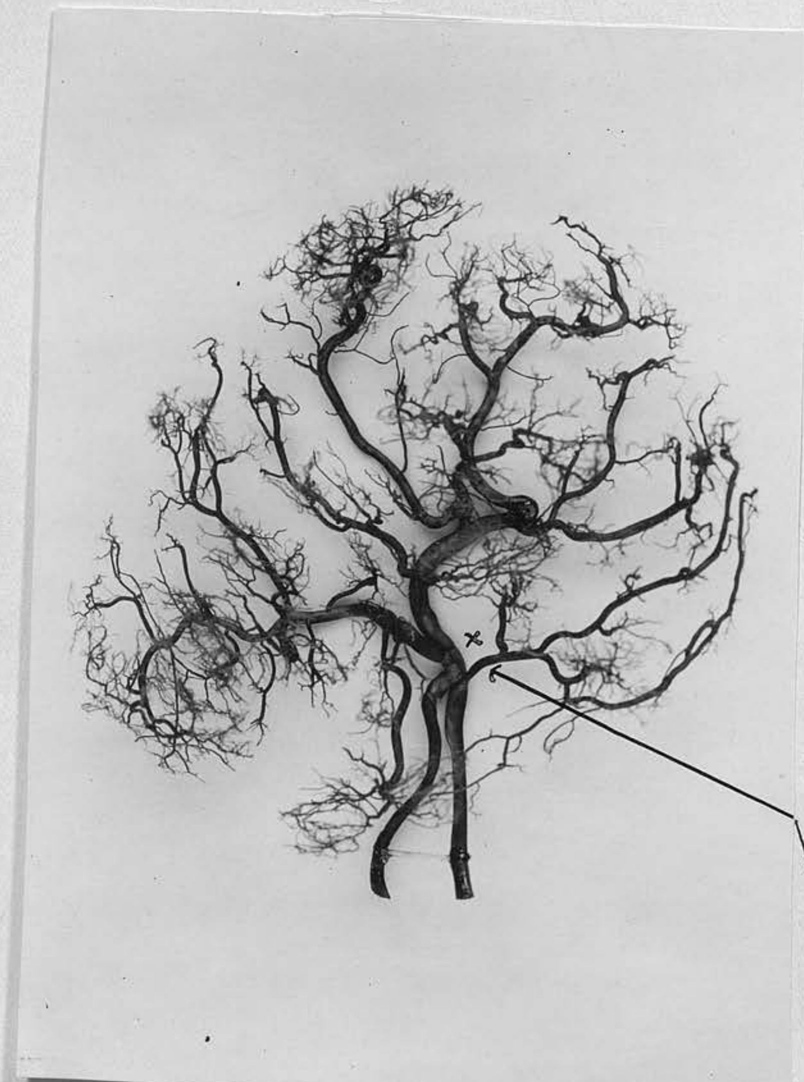
the/

the specimen left for 12-24 hours in the acid, not longer as prolonged immersion tends to render the celloidin more friable. It was found in the present investigation, especially with intestinal specimens, that immediate immersion in pure acid produced shrinkage of the specimen, with distortion of the vessels, tortuosity being produced as the organ shrank. Better results were obtained by prolonged immersion in 50% HCl for at least ten days. After that time, the specimen was removed, and washed under a gentle stream of water. The superficial, more macerated layers of the organ were thus washed away, exposing the deeper, less macerated areas to the acid. Repeated immersion followed, and repeated washing, till a completely clear cast was obtained. Gentleness in handling and patience were all-important here. Several immersions and washings, up to 4 - 5 weeks in 50% HCl, produced much better casts than with the pure acid. With placentae the strength of the acid used was increased to 75% on account of the fleshy nature of their structure. After washing, the specimen is ready for investigation. It forms a complete cast of the arteries of the organ, and is very suitable for/

for photography. If one of the main vessels has broken, it can be well repaired by a small drop of plastic "Durofix". Pettigrew (1934) mounted his celloidin specimens in a fluid medium:-

Formal 1%, Glycerine 10%, in distilled water. It has been found quite satisfactory in the present investigation to mount the specimens dry, and keep them in cardboard boxes. The casts made in 1939 are in as satisfactory a condition today (1946) as seven years ago.

Corrosion Cast of Placental arteries



Branch coming off just before junction of the 2 umbilical arteries.

The 2 umbilical arteries are held separated by a glass rod. The anastomotic channel takes the form of a fusion of the 2 vessels at the place marked "X". Note one branch comes off before this junction. There are no peripheral anastomoses

No.1 Photographs

Placenta (1). Corrosion cast of placenta. Photographed. The specimen shows the anastomotic channel between the two umbilical arteries at the entrance to the placenta. It shows also the branching of the arteries, running to the terminal cotyledons, without any peripheral anastomoses.

Placenta (2). Corrosion cast of placenta.

Owing to the failure of firm filling of the main trunks, this specimen broke up after corrosion into the separate branches of vessels of each cotyledon. The fine terminal ramifications were well seen without any anastomosis between the contiguous cotyledons. The different areas supplied by the two umbilical arteries were not distinctly visualised, as the red dye had perfused through before the blue dye was introduced. This demonstrated well therefore that the anastomotic channel between the two umbilical arteries at their entrance to the placenta allows blood to flow equally from one to the other.

No.2 Photograph (see next page)

Placenta (3). Corrosion cast of placenta.

The areas supplied by the two umbilical arteries are clearly seen on the actual specimen stained in red and blue. No anastomosis at all was seen distal to the connecting channel which had been ligated.

The photograph shows the cotyledons of the alkanin stained artery darker than the trypan blue stained vessels, and the fine terminal twigs, ending freely, without anastomoses.

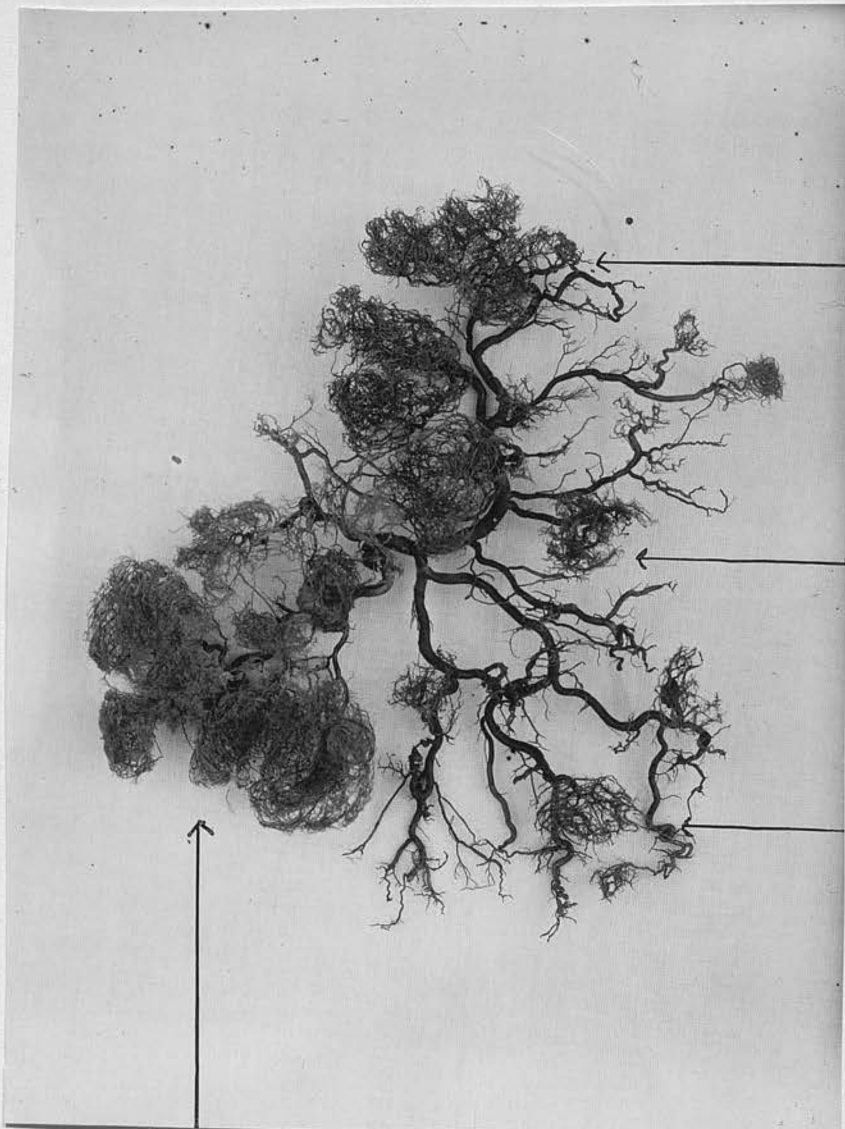
Placenta (4). Fresh specimen 8.3.46. Washed out with acidulated water. This specimen was injected with a mixture of vermilion, according to Ferguson's (1925) formula for roentological injection. This is made up as follows:-

Pale English Vermilion	dr. 2
Glycerine Amyli B.P.	dr. 3
Liniment Terebinth B.P.	dr. 1¼
Pulv. Tragacanth Co B.P.	gr. 1
Pulv. Amyli B.P.	gr. 3
Water	dr. 1½

A quantity of injection mixture was made up according to this formula using ordinary red vermilion as substitute for the Pale English variety. This was injected as Ferguson described, injecting by direct pressure with an all metal syringe, slowly, with the specimen floating in water to avoid injection/

no 2

Corrosion cast of Placental arteries



cotyledons supplied by artery injected with alkanin.

no peripheral anastomoses present.

cotyledons supplied by artery injected with trypan blue.

injection being prevented going into a part lying in contact with a supporting bench. The specimen was then X-rayed.

A patchy radiogram was obtained, the vessels presenting a broken up appearance. This was due to insufficient homogenicity of the vermilion suspension, the particles of the dye not being distributed uniformly throughout the mass. The radiogram showed, however, the two main umbilical arteries well, with the connecting anastomotic channel between.

No. 3 Photograph/

no 3

Corrosion cast of Placental arteries.

note fine terminal ramifications
of umbilical vessels, forming circular
meshes of vessels in the cotyledons. There are
no communications with other cotyledons.

No.3 Photograph

Placenta 5. Corrosion cast of placenta.

One umbilical artery was injected in the cord, the other being occluded by a pressure forceps in the cord.

Photographed. Both umbilical arteries had been filled, the celloidin flowing from the injected vessel into the other via the anastomotic channel. This specimen gave an excellent demonstration of the finest ramifications of the umbilical vessels, forming circular meshes of vessels in the cotyledons.

No anastomoses were visible peripheral to the main trunks.

Conclusions/

Conclusions

These experiments confirmed the results described by Bacsich and Smout (1938). The communicating channel between the two umbilical arteries at the entrance to the placenta was demonstrated in the five placentae studied. Beyond this main anastomoses the umbilical arteries break up into branches which go to separate cotyledons of the placenta. No peripheral arterial anastomosis is demonstrated either between the two umbilical arteries or between terminal vessels of each artery. The vessels of the cotyledons form separate distinct bunches of single vessels, each without any connection with its neighbours.

This arrangement of vessels,

(1) The communication between the main vessels,

and

(2) The solitary course pursued by the branches thereafter,

is admirably adopted to suit the requirements of an organ such as the placenta.

"The transverse communicating branch establishes an equal distribution of blood, and regulates the pressure in the placenta, counter-acting/

counteracting as a buffer system the effects of uterine contractions".
An anastomotic channel at the peripheral end would be useless in such an organ as it would be squeezed by the regular contractions of the uterus.

The branches of the umbilical arteries are par excellence examples of end-arteries. This is the ideal anatomical formation to suit the phenomenon of normal separation of the placenta. This is essentially a degeneration of the external (maternal) surface of the placenta, the plasmodium covering the villi degenerating into "fibrinoid" of which large masses may thus be produced (Queen Charlottes Text-Book of Midwifery). Such masses are frequently termed white infarcts, and the process of infarction by which the placenta separates is the natural sequence in an organ whose vessels are end-arteries.

The vessels of the placenta then exactly suit the function of this organ.

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SECTION II

The Pulmonary - Bronchial Anastomosis

The existence of an arterial anastomosis between the pulmonary and the bronchial arteries in Man has been the subject of much investigation. The bronchial vessels supply according to Ewart (1889), in addition to the air tubes

(1) The lymphatic glands and alveolar tissue at the root of the lung

(2) The pleura pulmonalis and the subpleural and interlobular tissue

(3) They are also believed to distribute nutrient blood to the pulmonary parenchyma, and vasa vasorum to the pulmonary vessels.

The pulmonary artery Ewart continues is exclusively devoted to the respiratory function, and unlike the majority of systemic arteries from its origin to its termination presents no anastomosis. At the extremity of the smaller bronchioles there is an important anastomosis between the <u>capillary</u> plexuses of the bronchial and of the pulmonary circulation. Note the word "<u>Capillary</u>".

W.S. Miller (1934) writes "Branches of the bronchial artery can be followed as far as the smaller bronchioli. With the appearance of alveoli along the walls of the bronchioli, the character of the/

the air tubes changes, and the bronchial arteries as a distinct set of vessels, disappear ..... up to the time of Guillot (1845) there was a general agreement that the bronchial artery anastomosed with the pulmonary artery. Guillot injected the pulmonary artery with a solution of gelatin coloured yellow with chromate of lead, and the systemic arteries through the aorta with a solution of gelatin coloured red with vermilion. After making more than thirty injections he did not find any evidence that the two systems anastomosed, but in every instance communications existed between the bronchial artery and the pulmonary vein". Miller made a series of five injections using the lungs of dogs. "Do anastomoses exist between the bronchial and pulmonary arteries? If the question be restricted to the arteries, it must be answered in the negative. If the question be asked if there be anastomoses between the bronchial and pulmonary circulatory systems, I answer in the affirmative; but it is by means of the capillaries". He points out a possible source of error due to fallacious interpretation "The vasa vasorum of the pulmonary artery are derived from bronchial arteries. A branch of the bronchial/

bronchial artery is traced into the wall of a pulmonary artery: therefore they anastomose". In a previous paper (1908) he noted another source of misinterpretation - applying findings in animals to human anatomy. "In the sheep and in the horse the type of the distribution of the bronchial artery to the pleura is different from that in man". "The question arises", he continues "what constitutes an anastomosis ..... (it is) a communication between two different sets of vessels ..... the absence of sizable vessels (in the communication) plays an important role in determining what constitutes an end artery". "In my study of the bronchial artery within the lung, I have failed to demonstrate by the use of granular injecting masses any anastomoses between the bronchial and pulmonary arteries ..... it is absurd to call an indirect capillary communication an anastomosis".

Ghoreyeb and Karsner (1913) wrote "In spite of the work of Miller, who claims that there is no real arterial anastomosis other than by terminal capillaries between the pulmonary and bronchial systems, the general opinion appears to be that functionally at least there is free exchange of blood, a dictum of Virchow practically contra-indicated in the earlier writings of Cohnheim and Litter/

Litter, who, however, retracted in later publications. In this period the work of Kuttner ..... supports the belief that there is extremely free interchange of blood, and rich anastomosis between the two systems. More recently Konigstein has made a series of studies of the comparative anatomy and physiology of the two systems in the lungs of reptiles, birds, and amphibia, and has reached the same conclusions".

"Kuttner showed that in the frog's lung the capillaries come off directly from relatively large vessels; that they are capable of taking over the function of anastomoses between two arteries or systems of arteries, that they can enlarge so as to resemble arterial anastomoses, and further that veins with the aid of capillary streams can function as anastomoses between two different arteries. Kuttner's work, however, is not without minor faults, and the later work of Miller would indicate that the intercommunication of the two systems is not as free as Kuttner believed." (Karsner and Ash, 1913). "With normal pressure in the two vascular systems (pulmonary and bronchial) an area of pulmonary embolism involving less than the entire lobe receives its blood supply almost entirely from/

from the rich anastomosis of the pulmonary artery between its own branches, and only when the bronchial pressure is raised to an extremely high point does the blood from this vessel play a notable part in the circulation in the area". (Karsner and Ghoreyeb, 1913).

Berry, Brailsford and Daly, (1932) investigating the bronchial vascular system in the dog stated "The more recent work (on this subject) tendsto show that the arterial connections between the pulmonary and bronchial systems are probably non-existent, but there is undoubtedly a capillary anastomosis ..... we have repeatedly attempted to demonstrate arterial communications between the pulmonary and bronchial vascular systems without success ..... there is no extensive arterial communication between the bronchial and pulmonary vascular systems in the dog, although our researches do not exclude the presence of the smallest arteriole twigs of the two systems".

In the guinea-pig, however, "it would appear that the probability of perfused blood passing from the pulmonary to the bronchial circulation, which is enhanced by a zero bronchial arterial pressure, is higher than in the dog, and is one/

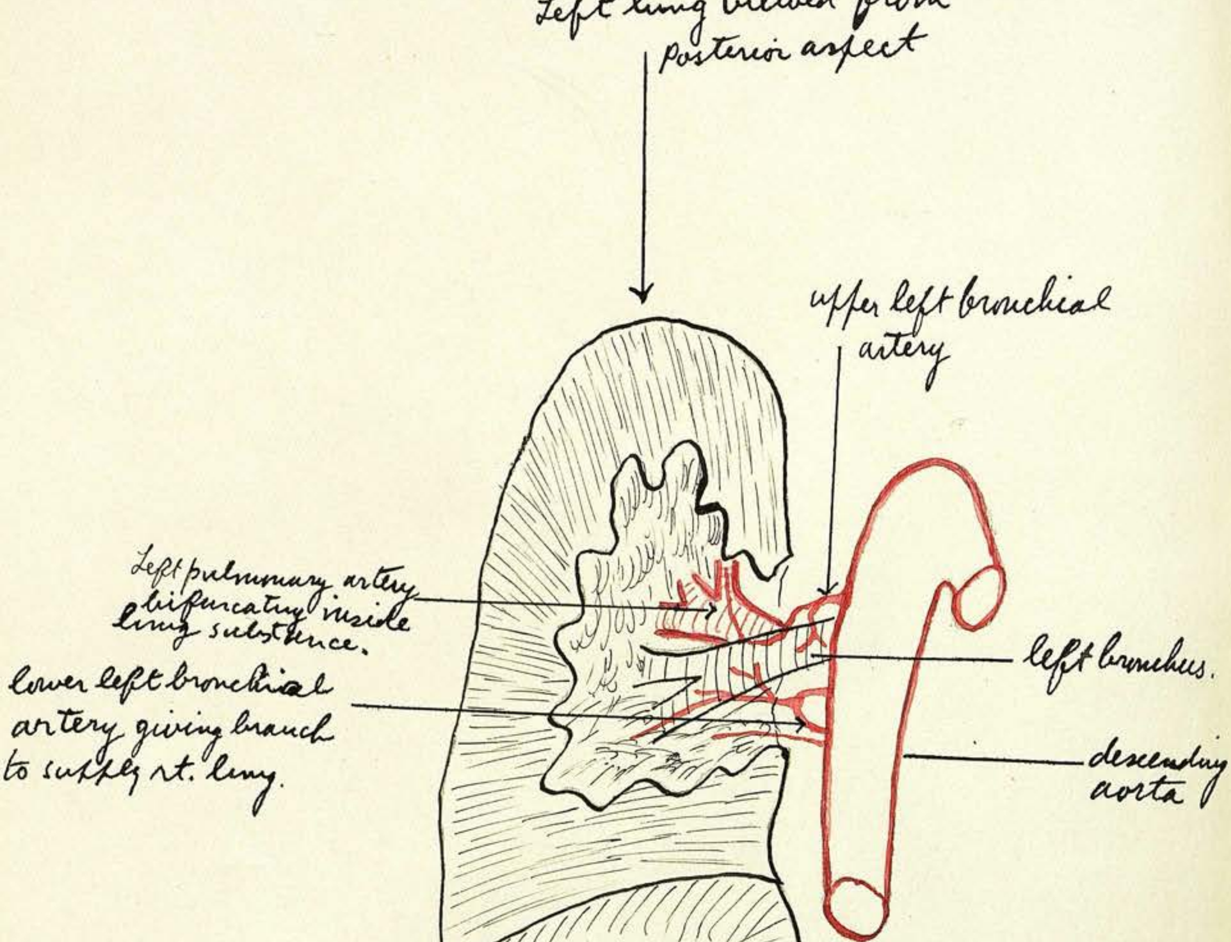
one which should receive further consideration" (Daly 1938). "The absence or presence of a "reverse" flow through the communicating vessels during simple perfusion of the pulmonary circulation may have an important influences on pulmonary response ..... drugs injected into the pulmonary circulation may also find their way into the bronchial circulation and to the tissues which it supplies, so that whether a "reverse" flow does or does not exist is of considerable importance in the assessment of the site of action of drugs injected into the pulmonary circulation" (Daly 1938). This is a method of mapping out the distribution of vessels, and their connections by "physiological" tests i.e. by injecting a drug into one artery and finding the phenomena due to its effect in the distribution of another, proving thus the presence of a connection between them. Daly points out the difference between the findings in the dog and in the guinea-pig, thus emphasising that findings in one animal cannot be applied to another without checking.

Somewhat conflicting views are therefore presented by different writers. The enlargement of bronchial arteries and their replacement to some extent of pulmonary artery function/

function, depends on the usual stimulus for enlargement of an artery-blockage of the other arteries of an organ. But this depends on the extent of the anastomotic channels between them. Potential channels must exist, to be available for enlargement when required. The existence of channels of any size, beyond the capillary size between pulmonary and bronchial arteries, was the point of controversy noted in the above review of previous work in this field, to form the subject of the present investigation.

Lung Experiments/

no 4


Left lung viewed from posterior aspect
upper left bronchial artery
Left pulmonary artery bifurcating inside lung substance.
left bronchus.
lower left bronchial artery giving branch to supply rt. lung.
descending aorta

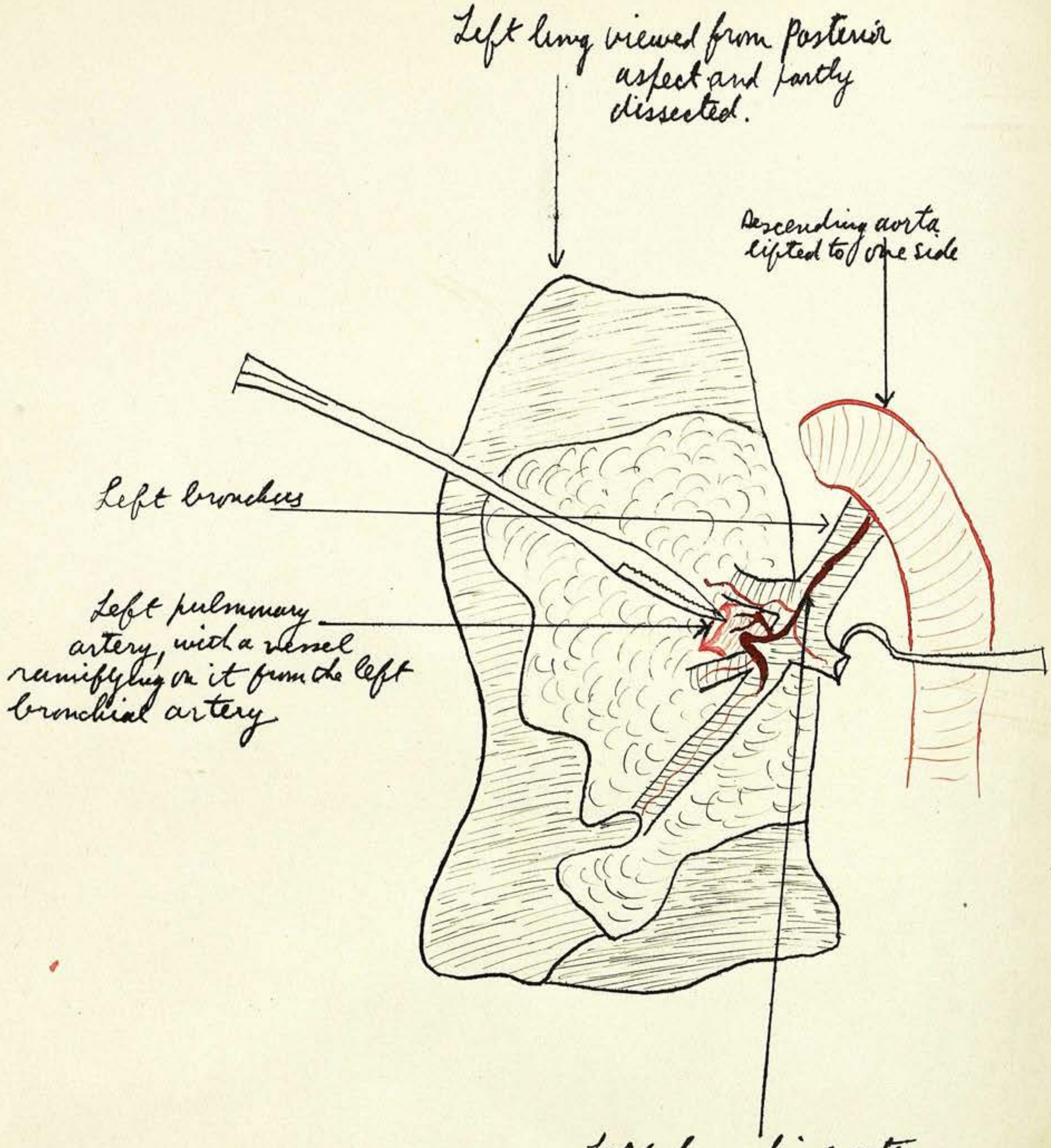
Lung Experiments

Nos. 1 - 6 preliminary experiments, see Appendix.

No.4 Drawing

Specimen 7. Left lung of full term foetus with aorta and pulmonary artery. Bronchial arteries injected via aorta.

The upper left bronchial artery is seen supplying obvious naked eye vasa vasorum to the left pulmonary artery's main trunk and branches, but no true anastomosis. This artery also supplies nutrient arteries to the trachea and oesophagus. The lower left bronchial gives off a branch which turns round to supply the right lung.


Left lung viewed from posterior aspect and partly dissected.
Descending aorta lifted to one side
Left bronchus
Left pulmonary artery, with a vessel ramifying on it from the left bronchial artery
Left bronchial artery

No.5 Drawing

(8) Lungs, trachea, aorta "pluck" of child aet 2½.

It shows the left bronchial artery (single) coming directly off the aorta and pursuing a course very closely applied to the bronchial stems, and having a kink at a bifurcation of the bronchi. A nutrient artery is seen going to the pulmonary artery (held by forceps).

(9) Full term still-born male infant, not breathed.

One right lung specimen.

A good cast of the pulmonary artery was obtained.

(10) Full term still-born male infant, not breathed.

"Pluck" of both lungs, trachea, oesophagus and thoracic aorta.

The specimen crumbled after maceration only the left lower lobe vessels remaining intact. Both pulmonary and bronchial arteries were showing, the bronchial artery lying as a fine thread loose on the pulmonary without any juncture.

(11) Full term still-born male infant, not breathed.

"Pluck" of both lungs, trachea, oesophagus and thoracic aorta.

Satisfactory cast of pulmonary artery obtained.

(12) Full term female infant, had breathed.

"Pluck" of both lungs, trachea, oesophagus and thoracic aorta. Unsuccessful.

(13) Full term male infant, not breathed.

"Pluck" of both lungs, trachea, oesophagus and thoracic aorta.

Successful casts of both lungs were obtained.

(14) Full term infant.

One lung specimen.

Pulmonary artery injected.

Result recorded as satisfactory.

(15) Full term still-born male infant.

"Pluck" of both lungs, trachea, oesophagus and thoracic aorta.

Satisfactory cast obtained of the pulmonary arteries, and the aorta with the bronchial arteries; no connection between pulmonary and bronchial casts visible.

(16) and (17). Full term still-born female infants.

After corrosion both the specimens broke into their right and left halves. A satisfactory cast of the left pulmonary artery was obtained, in both (16) and (17).

(18)/

Cast of right pulmonary artery of a full term still born foetus, viewed from in front. Glass rod for mounting fixed in main pulmonary trunk.

No.6 (Photograph)

(18) Full term still-born male infant, not breathed.

Specimen right lung.

Right pulmonary artery injected. Usual technique.

Good cast obtained. The photograph shows this cast with a glass rod for mounting fixed in the main pulmonary trunk.

(19) Full term still-born male infant.

"Pluck" of both lungs, trachea, oesophagus, and thoracic aorta.

Injected usual technique.

Good cast obtained of the pulmonary arteries of both lungs, with the aorta and bronchial arteries also lying loose and coming away separately after corrosion being able to be moved freely within the pulmonary vessel casts. No connection between the pulmonary and the bronchial systems demonstrated.

(20) Full term still-born male infant.

"Pluck" of both lungs, trachea, oesophagus and thoracic aorta.

A perfect cast was obtained after corrosion, the bronchial arteries coming off the aorta, passed laterally among the ramifications of the pulmonary arteries of both lungs, diminishing in size to fine threads just visible to the naked eye. They were not connected to the pulmonary vessels, the red bronchial vessel casts lying free among the blue pulmonary vessel casts. This specimen, like the preceding ones, was mounted dry. The cannulae were removed and a glass rod inserted into the pulmonary trunk, into the cavity produced by removal of the cannula, and fixed upright on a glass slab by a block of plasticine.

CONCLUSIONS

These twenty experiments were the nucleus of a wider and more extensive investigation which was commenced in 1939 with Dr. A.M. Macdonald, working in the Pathological Department of the University of Edinburgh; this investigation has not been continued following the interruption of the War. Unfortunately the casts of Nos. (9), (11), (13), (14), (15), (16), (19) and (20) have become missing during the war years, before being photographed; No. (18) alone remains.

Nos. (7), (8), (10), (15), (19) and (20) all demonstrated the pulmonary and the bronchial arteries. Nos. (7) and (8) were dissected. Nos. (10), (13), (15), (19), showed both systems in the same colour, and No. (20), the last, showed the pulmonary arteries in blue, the bronchial in red.

In Nos. (7) and (8), the bronchial arteries were demonstrated clearly as simple vasa vasorum to the pulmonary arteries' walls. In Nos. (10), (15), (19) and (20), the cast of a bronchial artery was found lying loose and free along the main stem/

stem of the pulmonary without any juncture, and could be moved by a gentle stream of water during maceration, and moved by forceps when dry.

With this celloidin cast technique, vessels down to the calibre of fine threads just visible to the naked eye were demonstrated. No anastomosis at this level was seen between pulmonary and bronchial arteries. The bronchial arteries in Nos. (7) and (8) were distributed to the wall of the pulmonary artery when this wall was macerated, as in succeeding corrosion specimens, the bronchial arteries lost their connection with the pulmonary, and the casts of the vessels were loose, one on top of the other. This would not have occurred if the lumens of the two systems communicated, the celloidin would then have flowed from one to the other, and after corrosion the connection would have become demonstrated as a continuity of cast.

This experimental work confirms the observations of Miller and Daly, detailed above, namely that the anastomosis between pulmonary and bronchial systems is in normal health a capillary one only, a type of anastomosis not demonstrable by the technique employed in the present work.

This/

This result accords with what one would expect to find - that is, no anastomosis between vessels of such unequal calibre as the pulmonary and the bronchial. Also, arterial anastomosis between arteries of such different functions as pulmonary and bronchial - the one respiratory the other nutrient-interstitial, would seem improbable, and does not, in the experiments detailed above, occur.

SECTION III

Alimentary Vessels

The blood supply of the alimentary tract is described by investigators in this subject with considerable diversity of opinion on many details.

Wilmer (1941) using the celloidin corrosion injection technique "as described by Hinman, Morison, Lee-Brown, Barker, Pettigrew" for investigating the first part of the duodenum, noted that "it is certain that variations are the rule in vascular surgery, and that the vessels to the duodenum are not constant and represent only a relatively constant pattern of pancreatic-duodenal arcades and anastomoses". This may explain the different accounts given by different authors.

Several papers have already been quoted in the Introduction, e.g. those of Hamilton Drummond (1914 a and b), Moynihan (1913), Stewart and Rankin (1933), Pope and Judd (1929) on the Colon, Eisberg (1925) and Cokkinis (1930) on the Small Intestine.

The/

The blood supply to the stomach and duodenum, with special reference to their relation to the etiology of peptic ulcer, was studied by Reeves (1919), some of whose investigations have been referred to in the Introduction. Reeves injected 62 human stomachs and duodenums, to determine if possible whether there is any difference in the character of the arteries in the stomach and duodenum in the regions in which ulcers are prone to occur, that is along the lesser curvature of the stomach, and on the anterior wall of the first inch of the duodenum.

He found "that all the arterial branches destined to supply the stomach penetrate the muscle coats and enter the submucosa where they form a very extensive plexus, or network of comparatively large vessels. Those from both curvatures anastomose freely with each other and reach across to anastomose with those of the opposite curvature. The plexus is remarkable in that all the vessels run a very tortuous wavy course and give off branches which are to a great extent of equal size throughout the entire stomach except along the lesser curvature. There the submucous plexus is made up of small perforating branches/

branches from the main trunks along the lesser curvature. On entering the submucosa, these vessels bifurcate and run more or less parallel with each other between the oesophageal opening and the pylorus. They are much smaller, make fewer anastomoses and run more than twice the distance of the same sized vessel in any other part of the stomach. By means of rather small branches, this plexus anastomoses with those on the anterior and posterior walls.

The first one and a half inches of the duodenum receive their blood supply chiefly from an artery which is usually given off from the gastro-duodenal or hepatic. This artery has been described at length by Wilkie under the name of 'supraduodenal artery'. It anastomoses rather sparingly with a small branch of the pyloric, a small branch of the right gastro-epiploic, and with branches of the superior pancreatico-duodenal. The posterior wall of the first one and a half inches of the duodenum is supplied chiefly by small branches from the gastro-duodenal artery, given off as that vessel passes behind the bowel. It also receives some small twigs from the supraduodenal, pyloric, and right gastro-epiploic arteries. These arteries/

arteries, soon after reaching the wall of the duodenum, penetrate the muscular coat and form a submucous plexus which is strikingly different from that lower down in the bowel. The first inch certainly has very few arteries in the submucosa in comparison with other parts of the duodenum. It would seem that this explains the observation of W.J. Mayo regarding the 'anaemic spot' produced by traction usually seen on the surface of the duodenum in this region". Reeves concludes that the anatomic arrangements of the arteries along the lesser curvature of the stomach are such that the arteries are predisposed to thrombosis; the tortuous, spiral courses of the branches from the submucous plexuses in these regions not straightening out as elsewhere in the stomach when the stomach fills. The submucous plexus of arteries in the first inch of the duodenum is made up of relatively few vessels in comparison with other parts of the duodenum. They are small and do not anastomose freely, they give off branches to the mucosa some of which stimulate the gastric type of spiral artery, thus predisposing to thrombosis and haematogenous emboli (and so produce the origin of a peptic ulcer).

Wilmer/

Wilmer (1941) investigated the blood supply of the first inch of the duodenum with special reference to the problem of the bleeding ulcer, and possible surgical relief of this condition. He found that the variations in pattern were so common, and the gastro-duodenal plexus so complex that it is unlikely that vessel ligation will suffice to control bleeding in massive haemorrhage from a posterior wall duodenal ulcer. These observations point to a different conclusion than Reeves and suggest that a cause other than vascular pattern must be sought for to explain the etiology of peptic ulcer.

The fact that with the exception of the pyloric end of the stomach, and the first portion of the duodenum the recto-sigmoid is more frequently diseased than any corresponding portion of the gastro-intestinal tract (Mayo 1917) has lead to extensive researches in the anatomy of this region, including its blood supply (e.g. Archibald 1908).

The blood supply of the colon has been described in detail by Fraser (1938) referring in particular to the work of Jean Meillere.

Jean/

Jean Meillere (1927) in his beautifully illustrated study of the vascularisation of the coats of the large intestine and its surgical application, noted the constant relationship of the vasa recta of the colon to the bases of the appendices epiploicae. "C'est sur le trajet des vaisseaux droit longs, et pres de la bandelette distale que sont implantés les appendices epiploiques". He stated, that in his injections, more often than not these vasa recta described a loop in the base of the appendix, five to ten mm. or even more from the intestine. He noted, also, if he injected after "disinsertion" of an appendix epiploica level with the surface of the colon, then the triangular zone with its base at the free border and its summit at the mesocolic border supplied by the underlying vasa recta was not injected. "La forme du territoire des vaisseaux long explique bien les zones de gangrene observées apres certain enterorraphies circulaires".

If this interesting observation of arterial loops in the appendices epiploicae confirmed a common, but little recognised formation, then one would expect to find in the literature on torsion of the/

the appendices epiploicae some reference to the fact that in these cases gangrene of portions of intestinal wall in relation to the appendices epiploicae sometimes occurred owing to inclusion of the arterial loop in the twisted appendix.

But a study of this literature does not reveal any such finding. V.C. Hunt (1919) described eleven cases of torsion and inflammation of appendices epiploicae, and gave references of forty-two more; he noted also that in four cadavers free bodies in the peritoneal cavity, thought to be dropped off appendices epiploicae, have been found (Littre 1703; Laveran 1895; Cruveilhier 1894; Virchow 1863).

In none of these cases is there any mention of gangrenous patches on the intestinal wall, and in a case described by him at operation, a sac, 4 x 3 x 3 cm., containing degenerated fat "probably an appendix epiploica which had become twisted from its attachment to the large bowel" was removed; the bowel was healthy.

Taylor (1931) Harrington (1917) Randall (1932) Colt (1932) Fiske (1936) Hartly (1922) describe cases of torsion of the appendices epiploicae, with uneventful recoveries following surgical removal of/

of the strangulated appendices; while Klingerstein's (1924) study of some phases of the pathology of the appendix epiploica makes no reference to basal arterial loops.

Here, again, then, we find variance between observations of different authorities.

A very extensive review of the blood supply of the large intestine is that of Stewart and Rankin (1933) who investigated eighty complete colons "from the ileo-caecal valve to the iliac colon or sigmoid". They found that the ileo-colic artery is a constant vessel, and that the right colic artery is the most inconstant of the colic arteries. In the specimens studied, the right colic artery originated from the superior mesenteric in 40% of cases, from the middle colic in 30%, and from the ileo-colic in 12%. In 18% there was no artery that corresponded in course or distribution to the right colic. The middle colic was variable; the left colic was present in every specimen, though its course and its branches were extremely variable. Though the feeders varied, the peripheral vessel they ran to was constant - a marginal artery was found regularly, running at varying distance from the wall of the colon, with secondary loops in different portions of its course. In more than 100 specimens studied/

studied there was no failure of anastomosis of the middle and left colic arteries.

Stewart and Rankin make the following remarks about the terminal arteries of the colon:-

1. They are of two types, long branches which supply the amesocolic or distal third of the colon wall, and short branches which supply the mesocolic or proximal two thirds of the colon wall.
2. The mesocolic taenia portion of the colon has most of the blood supply.
3. The course of the terminal arteries is in general perpendicular to the axis of the bowel.
4. There is little anastomosis between the terminal vessels except in the submucosa.

They do not note any marked difference in the arterial patterns between the left and the right halves of the colon, apart from stating "Terminal arteries are more numerous in the caecum and ascending colon".

The blood supply of the caecum is discussed by J.R. Cameron (1939) in his study of simple non-specific caecal ulcer. Caecal ulcers are situated "on a level approximately with the ileo-caecal valve. In this they bear an anatomical as well as pathological relationship to ulcers of the duodenum. In both, the ulcer develops opposite to a segment of gut guarded by a valve or sphincter having a similar and connected innervation; in both the/

the blood supply of the affected area is relatively deficient, particularly when the bowel is distended, the vasa brevia and longa (anterior and posterior caecal arteries) terminating or inosculating with little anastomoses as does the artery supplying the anaemic spot on the anterior duodenal wall".

Cameron, however, was able to find only 23 cases of simple non-specific caecal ulcer including his own, recorded in the literature. It is reasonable to suppose that if there were any marked peculiarity or deficiency of the blood supply of the caecum, then caecal ulcer would be vastly more common than it is, the caecum being an organ specially liable to distension and stasis of its contents.

Barron (1928) reviewing 53 cases of simple non-specific ulcer of the colon makes no special reference to peculiarities of blood supply to the colon, beyond discussing the effect of constipation on the colonic circulation. This "produces distension; as a result of this distension there is a diminution in the arterial blood flow soon after distension occurs. The intestine becomes pale and ulcers may develop even at this stage".

Noer/

Noer (1943) has investigated the blood supply of the jejunum and ileum, a comparative study of man and certain laboratory animals. He prepared his specimens by means of liquid latex injections as described by Batson (1939), because celloidin corrosion fails to show the relationship between the vessels and the intestinal wall.

He found in man "considerable variation present in the manner by which the vasa recta reach the intestinal wall; they may pass directly to one side or the other of the intestine, whereas others divide before reaching the mesenteric border, and send branches to both sides. Still others divide but send all their branches to one side only; there is no definite pattern characteristic of any given level, the variations being distributed in a haphazard manner .....

The human mesenteric vascular pattern is one of multiple mesenteric arcades increasing in number distally. Vasa recta pass from these arcades to the intestinal wall without any intercommunication and then course to either or both sides of the intestines as mural trunks ..... There are three types of antimesenteric anastomoses:

(1) direct communications
(2) plexiform type
(3) transverse vessels joining arcuate mural anastomoses".

Another/

Another feature, the distance from the mesenteric attachment at which vessels pierce the muscularis he considered to be a factor which has been overemphasized. He found no significant differences between the upper jejunum and the lower ileum. "Vessels tend to pierce the muscularis in the mesenteric half of the intestine, but within the area the variations appear to be great and to occur at random".

In the dog he found the vascular pattern varies little throughout the jejunum and the ileum. It consists of large primary and secondary arcades which lie immediately adjacent to the intestinal wall, with short and abundantly intercommunicating vasa recta passing between the peripheral arcades and the intestinal wall. "The mural trunks intercommunicate freely by direct or by plexiform anastomoses ..... there is a well developed secondary network of fine vessels lying near the serosal surface".

Following a perusal of the literature, experiments were performed to determine some/

some of the controversial points raised in the study of arterial pattern in the alimentary tract.

Segments of (1) The human colon

and

(2) the human small intestine were studied,

and also

the small intestine of a dog, for comparison.

Colon (1). 14½ inches of transverse colon with attached transversed mesocolon.

This specimen showed a well defined marginal artery, a single trunk. Off this came vasa recta longa and brevia; the vasa recta longa to alternate sides of the colon, running sub-peritoneally to the amesocolic taeniae where they dipped underneath these muscle strands. Arcade formed at one place by a channel across the bifurcation of the middle colic artery.

Twenty-two appendices epiploicae were detected on this specimen, between the amesocolic taeniae. They were in the form of single tags, and broader folds with fimbriated ends. In no case was there any arterial loop detected in their base, produced by a vas rectum.

Colon (2) Female aged 74. Died of carcinoma of gall-bladder, with gross obstructive jaundice.

Specimen terminal ileum, caecum, and 48 inches of ascending, transverse descending and iliac colon.

Abundant fat present extending in ridges from the mesocolic attachment to the anti-mesocolic border, "flagged" by the appendices epiploicae.

Numerous/

Numerous large appendices epiploicae present, in a double row, one on either side of the intestine, till the iliac portion, where a single row was present only on the anteromedial aspect.

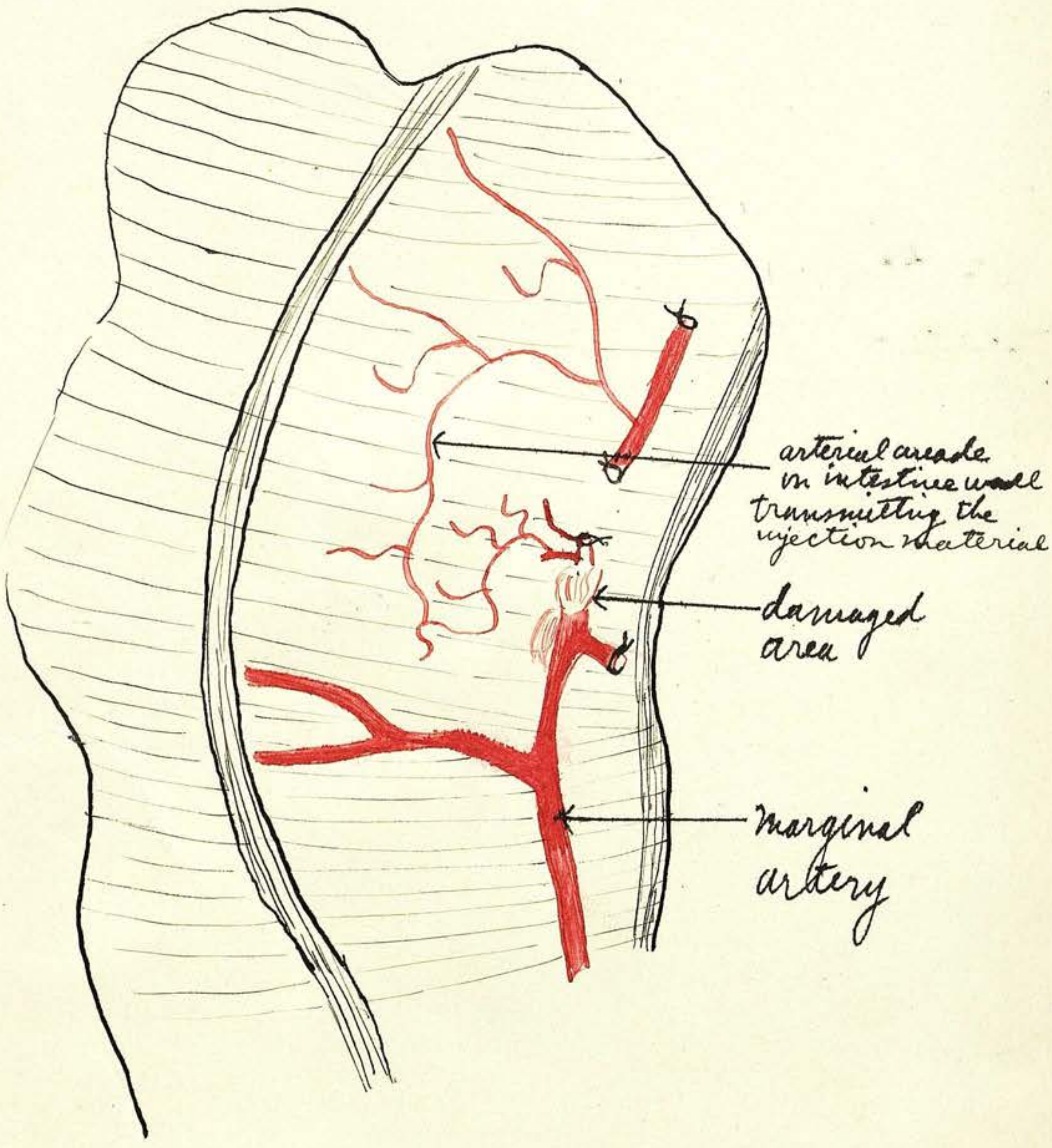
The main arteries ileocolic, right colic, mid colic, left colic and upper sigmoid were well injected. A well developed complete single "marginal" artery was present all round from the ascending branch of the left colic artery to the lower end of the iliac portion of the colon. This single longitudinal channel was produced by the bifurcation of the main arteries, and the anastomosic channels across the bifurcation. Coming off the marginal artery were the vasa longa and vasa brevia. These had an irregular origin coming off (1) as single trunks to alternate sides of the colon (2) as larger vessels running to the margin of the gut, and there dividing into two, one to each side of the colon (3) as single arteries running to the colon and there forking into two parallel vessels running to the same side of the colon. These vessels ran slightly sinuous courses, in grooves covered by the fat ridges which were noted above. In two places, towards the distal end of the specimen, there was a shallow loop in the vas rectum at the base of an appendix epiploica.

As in (1), the vessels were noted to pierce the colon just proximal to the taeniae coli.

<u>No. 7 (Drawing)/</u>

No 4

Segment of transverse colon.



No. 7 (Drawing)

Colon (3). Male aged 44 died of diabetes.
Specimen - portion of right colic flexure and transverse colon 19 inches long, with the great omentum. Thin subject.

A well defined marginal artery was present, 1/3 inch from the colon margin; formed by the divisions of the main feeding vessels. Right and middle (2) colic arteries, with the arcade anastomoses across the points of bifurcation. ½ inch from the injection point the main marginal artery was damaged, but the injection flowed through into the marginal artery beyond the damaged area by the arcade anastomosis across the bifurcation and was distributed to sixteen main vasa longa which came off on to the anterior surface of the colon. They arose alternately with those on the other side of the colon which had fifteen. These origins were variable. Some came off singly, some came off a common stem which then bifurcated, the vessels running to the grooves between the sacculations of the colon, piercing the wall gradually proximal to the taeniae coli.

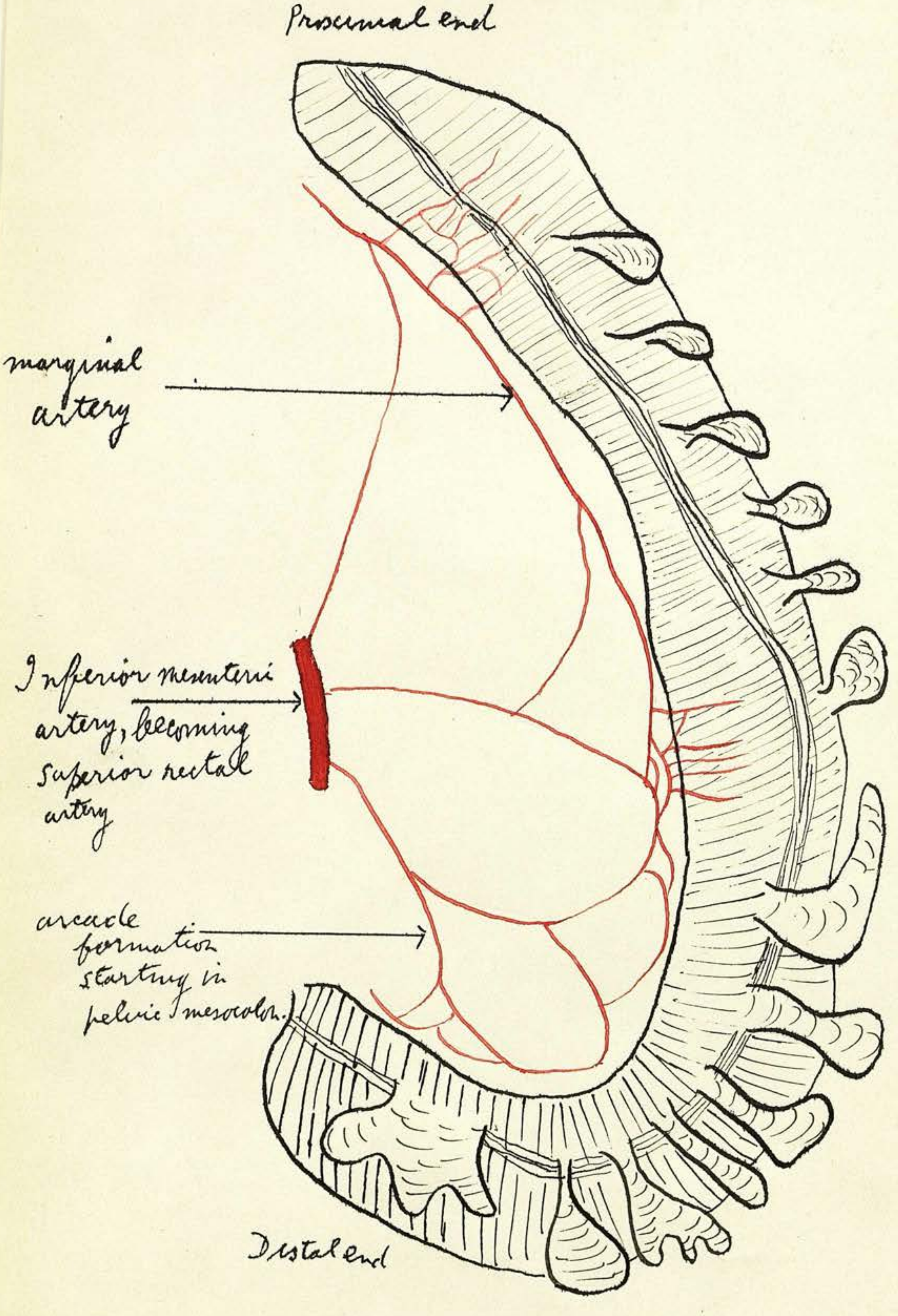
A single row of appendices epiploicae was present on the anterior aspect, the appendices being set on the course of the vasa recta. In several of these appendices the vascular supply was seen. This consisted of one or two slender longitudinal vessels, coming off the vasa longa, and running to the tip of the appendix. There was no loop in the vas longum in the base of any appendix; the vasa longa pursued straight courses, or with wide sinuous bends, but no true loops.

Numerous vasa brevia came directly off the marginal artery, and off the vasa longa. Some ran parallel to the vasa longa, i,e. at right angles to the longitudinal diameter of the gut, others branched off the vasa longa in line with the longitudinal diameter.

After dissecting off the peritoneum and taeniae coli, the vasa longa were traced as far as their terminations. One anastomosis was present in this specimen, between two vasa longa. No anastomosis across the mid line was detected.

No. 8 (Drawing)/

Loop of descending, iliac and upper pelvic colon.



No. 8 (Drawing)

Colon (4) Female aged 56. Died following decompression for cerebral tumour.

Specimen - 20 inches of descending iliac and (upper) pelvic colons.

The inferior mesenteric artery, giving off three sigmoids, and continuing as the superior rectal, was demonstrated. These vessels ran to the colon, and communicated by an arcade anastomosis system at the border of the intestine, a continuous marginal artery being thus formed. In the iliac portion the marginal artery so formed was large, and closely applied to the margin of the colon. In the inferior pelvic portion the third sigmoid artery bifurcated, and the branches so formed bifurcated again with anastomotic arcades across the bifurcations, forming a plexiform arrangement rather than a single trunk. Fine anastomotic channels across between vasa longa on the colonic wall, were present also in places.

The vasa longa arose at regular intervals, singly or two forking from a common stem to run to either side of the colon, or parallel with each other on the same side. The vasa longa were all of an approximate callibre. The vasa brevia were also of a similar calibre; some vessels intermediate in size between longa and brevia were present.

Traced through the muscle wall, the vasa longa were found to reach the submucous level, but there the injection stopped abruptly, and there was no complete encirclage of the intestine.

Two well defined rows of appendices epiploicae were present, the larger appendices being on the anterior aspect proximal to the anterior taenia coli, the appendices on the posterior aspect being much smaller. Without exception these appendices were on the course of a vas longum. Thirty-two were counted on the anterior aspect.

The vasa longa ran in ridges of fat to the bases of the appendices epiploicae; it was possible to lift them in this fat 10 - 12 mm off the actual gut wall. From them the vasa brevia came off practically vertically to the gut wall.

No. 9 (Drawing)/

No. 9 (Drawing)

The vessels of the appendices epiploicae came off the vasa longa, running from the centre to the tip; some had single vessels, several had three. There was no loop formation of the vas longum, except in one instance where an artery with a sinuous course had a wide curve in the base of an appendix epiploicae.

Colon (5). Male aged 21. Died of fractured skull.

Specimen - 21 inches of descending, iliac and pelvic colon.

A well defined marginal artery was present in the upper part of the specimen. In the mesocolon an arcade formation was present, the marginal artery being formed here by the anastomotic channel across the bifurcation.

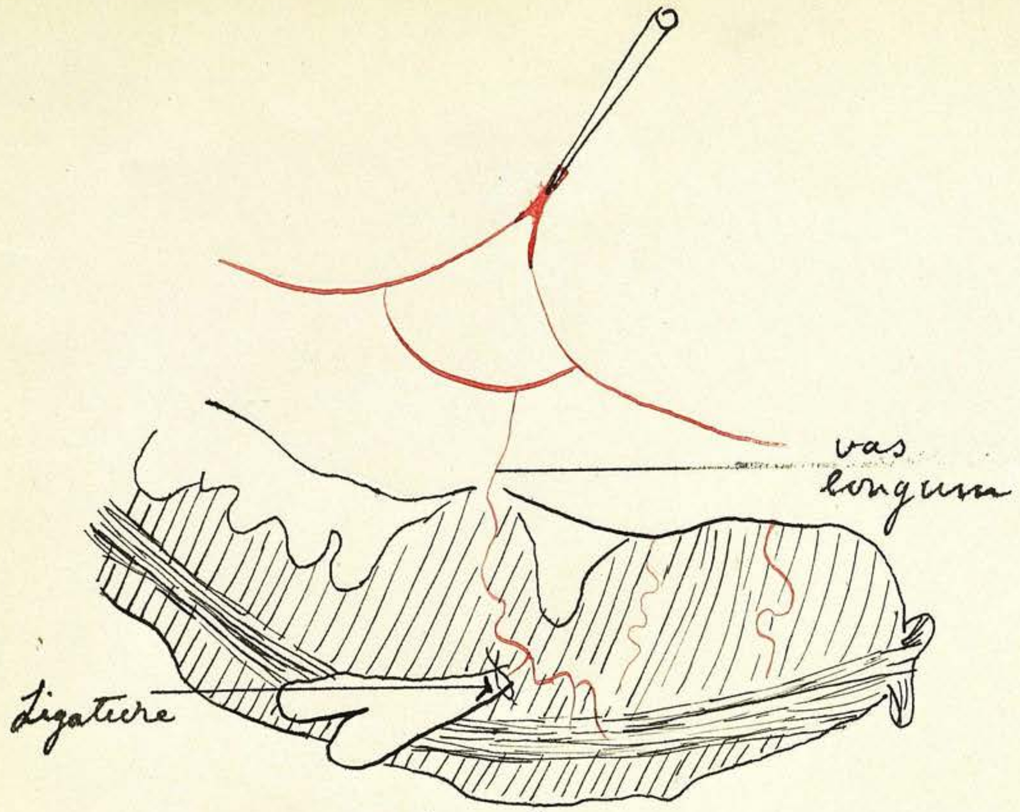
The vasa longa came off the marginal artery at right angles, and began to pierce the muscle coat just proximal to the anterior taenia coli. The vasa brevia ran in two directions

(1) slender vessels parallel with the vasa longa,
and (2) shorter vessels branching off the vasa longa on the gut wall.

The vasa longa came off the marginal artery
(1) alternately to opposite sides of the colon
(2) together at the same level from the marginal artery
(3) by a short common stem which then bifurcated to either side of the colon.

The vasa longa were "flag-marked" in their course by appendices epiploicae, which were present in two rows, a row of larger ones anteriorly, and a row of smaller ones posteriorly. The appendices epiploicae received a slender median vessel coming off the vasa longa at right angles. In no instance in 26 appendices examined was any loop demonstrated in root of the appendices epiploicae, produced by the vasa longa. An imperfect system of anastomoses between the vasa longa on the colon wall appeared in this specimen. Five such vessels being present connecting the vasa longa, produced by transverse running vasa brevia meeting. From their union a small vessel ran towards the taenia coli. The distance between the vasa longa where these transverse anastomoses occurred did not differ from the distance between vasa longa where there were no anastomoses. In one place an anastomosis was formed by two vasa longa curving towards each other joining near the taenia coli, and from this four vasa brevia arose.

No anastomoses was formed between vessels from opposite side of the colon at the anti mesocolic border.



segment of colon.
Ligature applied round base of an appendix
epiploica, and injection material flowed beyond
it, without being obstructed by the ligature.

No. 10 (Drawing)

Colon (6) Male aged 60. Died of uraemia due to chronic interstitial nephritis.

Specimen - eight inches of pelvic colon.

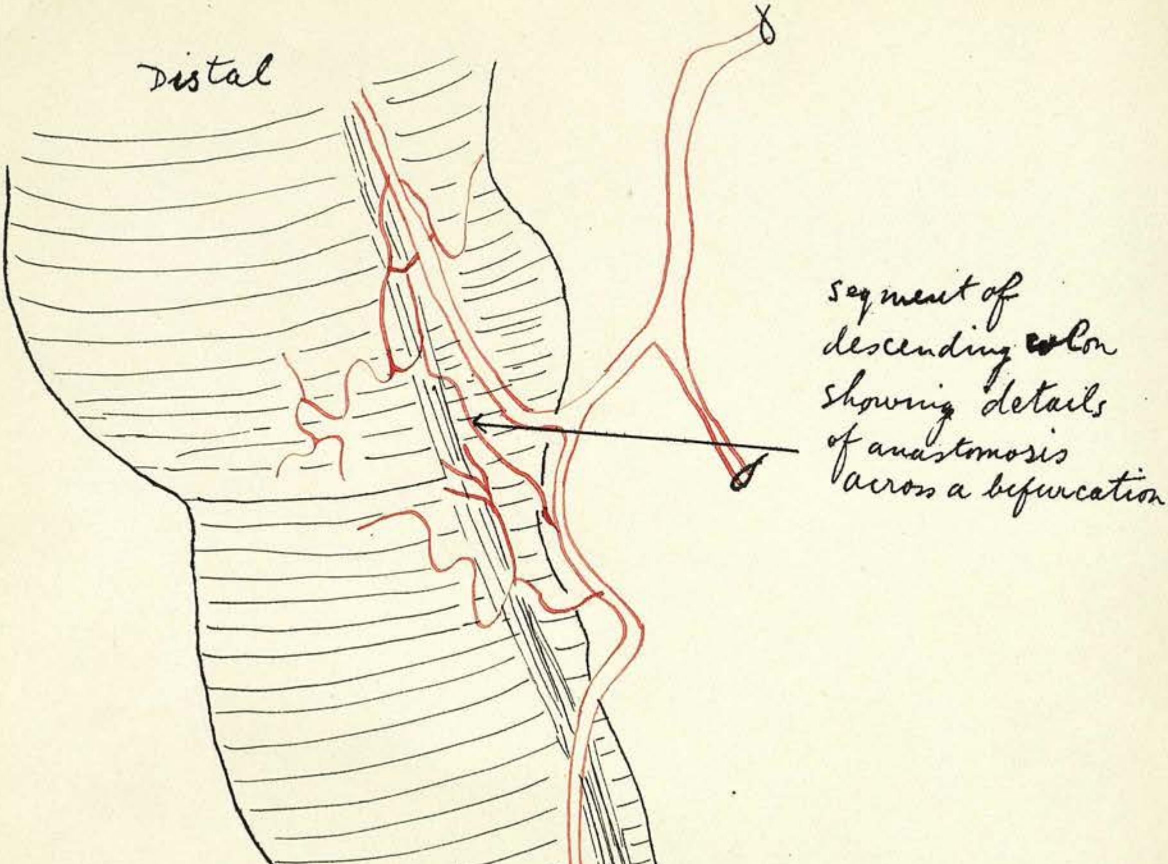
A marginal artery was present; the vasa longa came off alternately to either side of the colon either directly or from a short common stem which bifurcated. The vessels pursued a sinuous course, being "flag-marked" by appendices epiploicae.

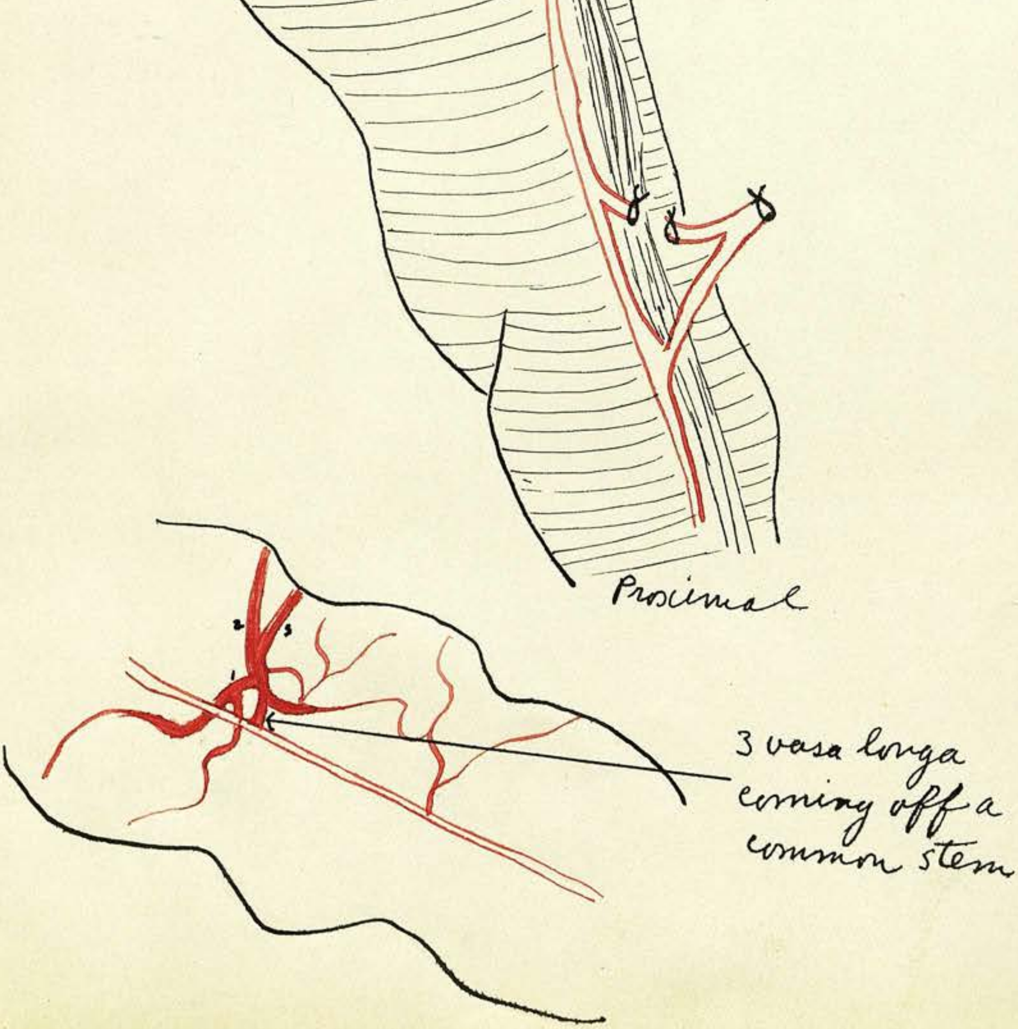
Prior to injection, one appendix epiploica was lifted up without any undue tension and ligated at the base. As seen in the drawing the injection continued to flow along the vas longum distal to this ligature.

No cross anastomosis was present at the anti mesenteric border.

No. 11 (Drawing)/

no 11


Distal
segment of
descending colon
showing details
of anastomosis
across a bifurcation
Proximal


1
2
3
3 vasa longa
coming off a
common stem

No. 11 (Drawing)

Colon (7) Male aged 60. Died of cerebellar tumour.

Specimen - 29 inches of descending and pelvic colon.

A well defined single marginal artery was present in the proximal part of the specimen, being replaced by the "primary" arcade in the distal half.

The vasa longa came off singly to alternate sides of the colon, or two off a common stem. In one instance three came off one common root. They ran in a tortuous fashion, piercing the muscle coat at the amesocolic taenia coli on either side. They were "flag-marked" by small appendices epiploicae, no loop being present in the base of the appendices epiploicae anywhere.

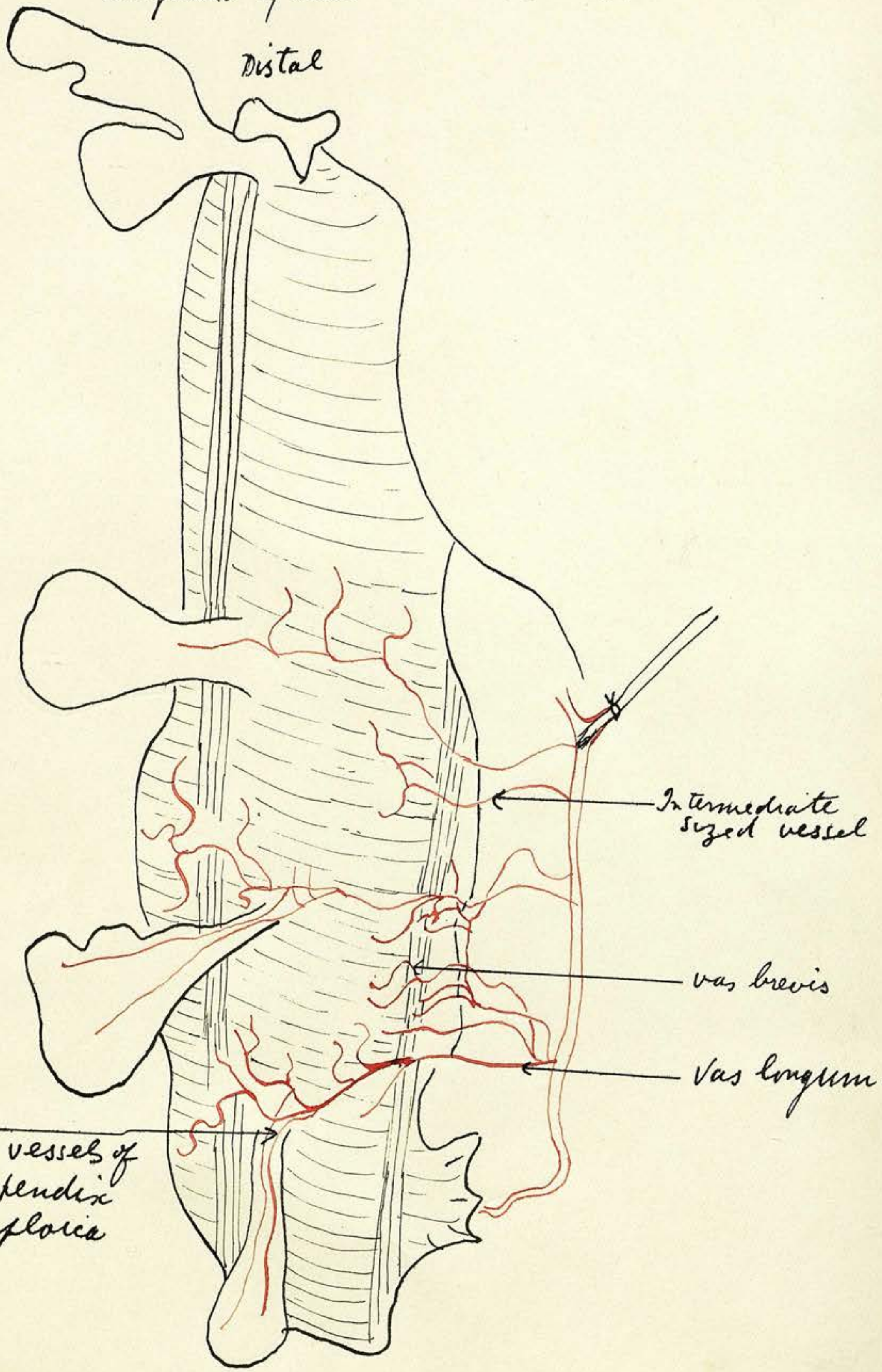
A series of secondary arcades was present, being better developed in the pelvic mesocolon of the lower part of the specimen, between the vasa longa. In several places a cross anastomoses was present between vasa longa of the opposite sides on the colon wall.

Vasa brevia came off the marginal artery parallel with the vasa longa and off the vasa longa themselves. An incomplete series of tertiary arcades was present between these vasa brevia on the gut wall.

No. 12 (Drawing)/

no 12

Segment of mid transverse colon.



No. 12. (Drawing)

Colon (8) Female aged 59. Died of carcinoma of head of pancreas.

Specimen - 7 inches of mid transverse colon.

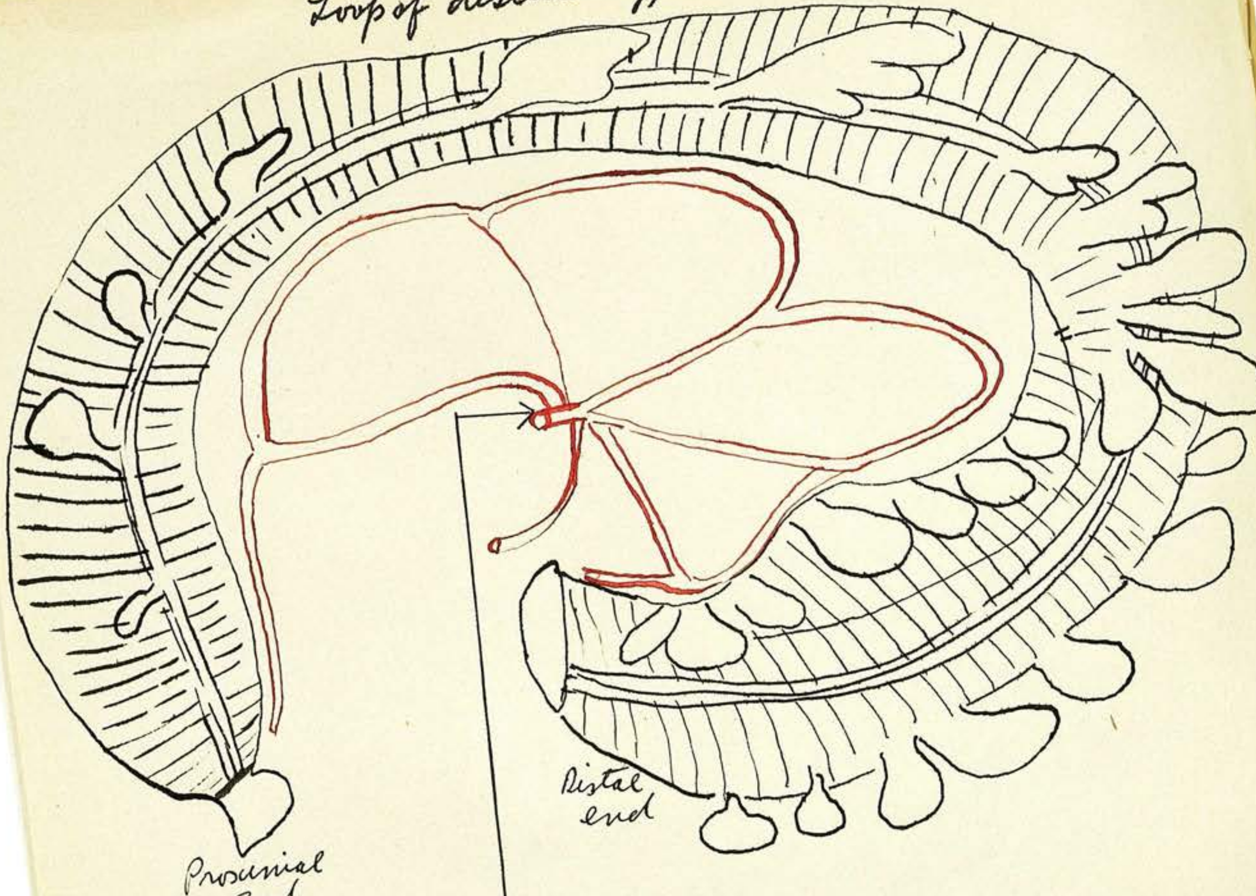
A marginal artery was present. The vasa longa were tortuous, but no actual loop in the bases of the appendices epiploicae was seen. The injection failed to reach the distal half owing to complete lack of anastomosis between the vasa longa. The vessels of the appendices epiploicae were well seen. The vasa brevia reached the gut directly off the marginal artery, or off the vasa longs. An intermediate vessel was also seen.

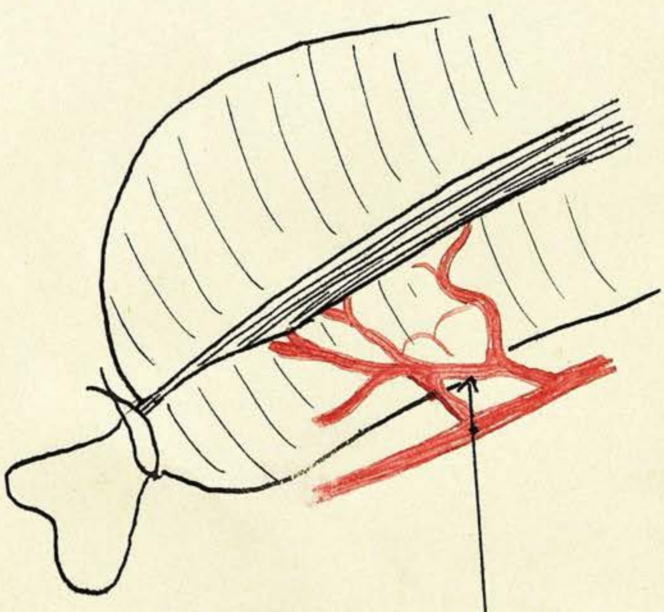
Secondary arcade formation was present at the margin of the colon.

Nos. 13, 14 and 15 (Drawings)/

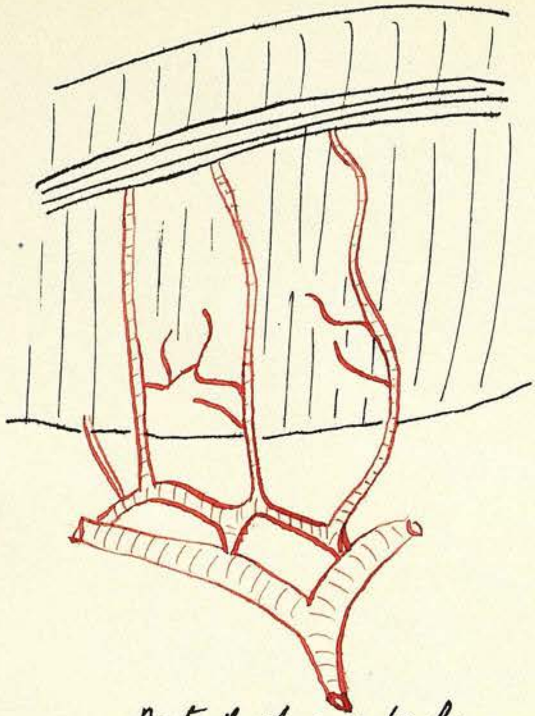
No 13

Loop of descending, iliac and pelvic colon

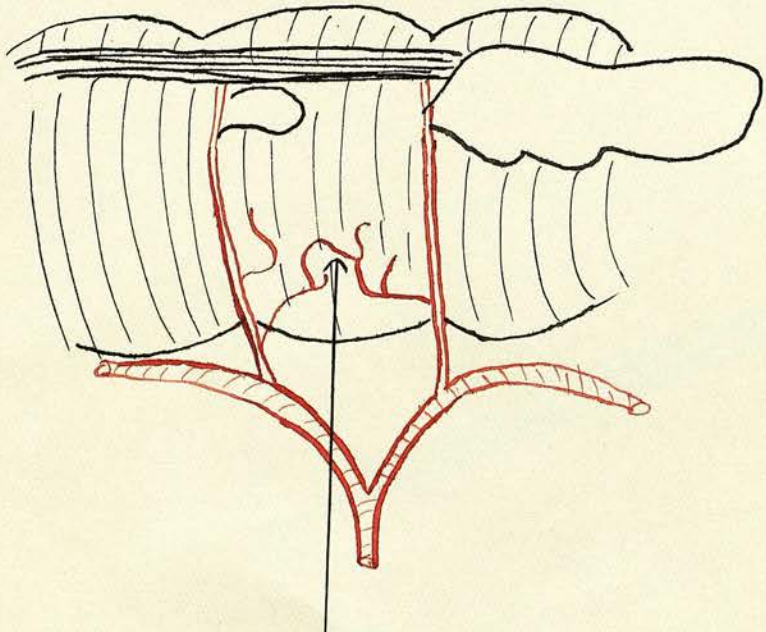




no 14

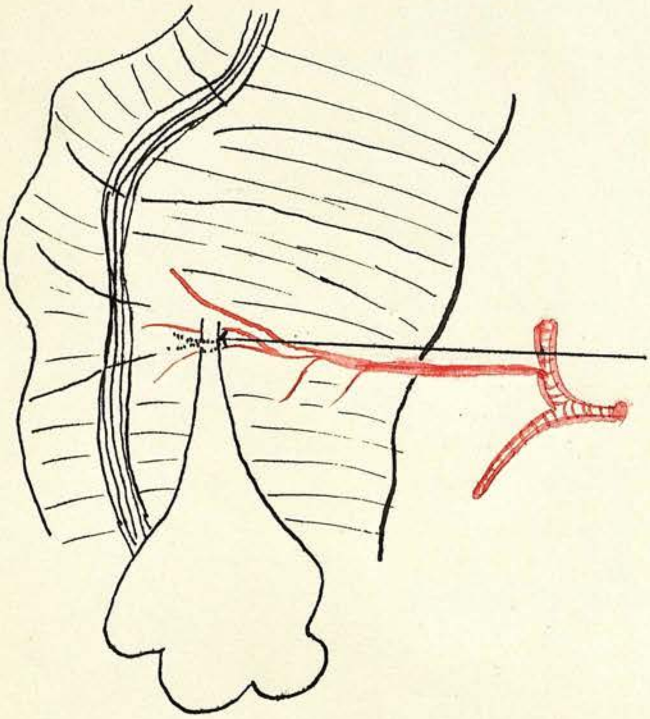


Details of arcade formation

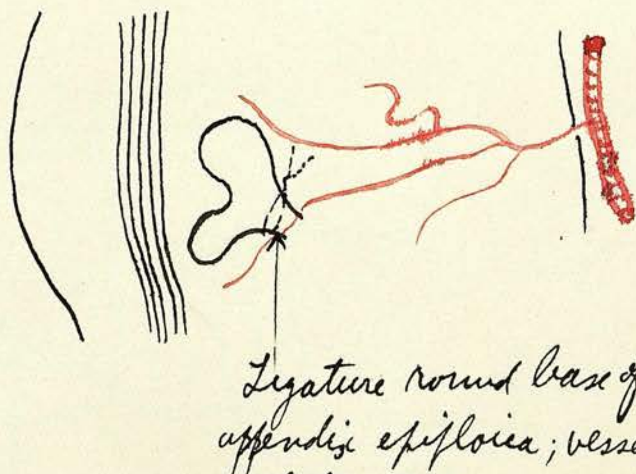


Pelvic colon. anastomosis on gut wall, across a bifurcation of main feeding vessels.

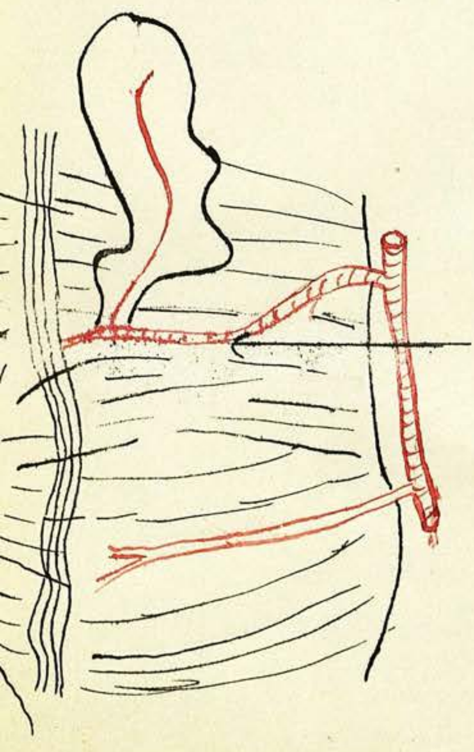
no 15



Ligature at base of appendix epiploica; flow of celloidin not interrupted into course of vas longum beyond the ligature



Ligature round base of an appendix epiploica; vessel not obstructed



vas longum.

Nos. 13, 14 and 15 (Drawings).

Colon (9) Female aged 55. Died of coronary thrombosis.

Specimen - 30 inches of descending iliac and pelvic colon.

A continuous marginal artery was present close to the mesocolic attachment of the gut, formed by anastomotic channels across the "primary" arcades produced by the branches of the inferior mesenteric artery.

The vasa longa came off the marginal artery

(1) alternately to alternate sides of the colon

(2) off a common stem, bifurcating to alternate sides

(3) off a common stem to the same side of the colon.

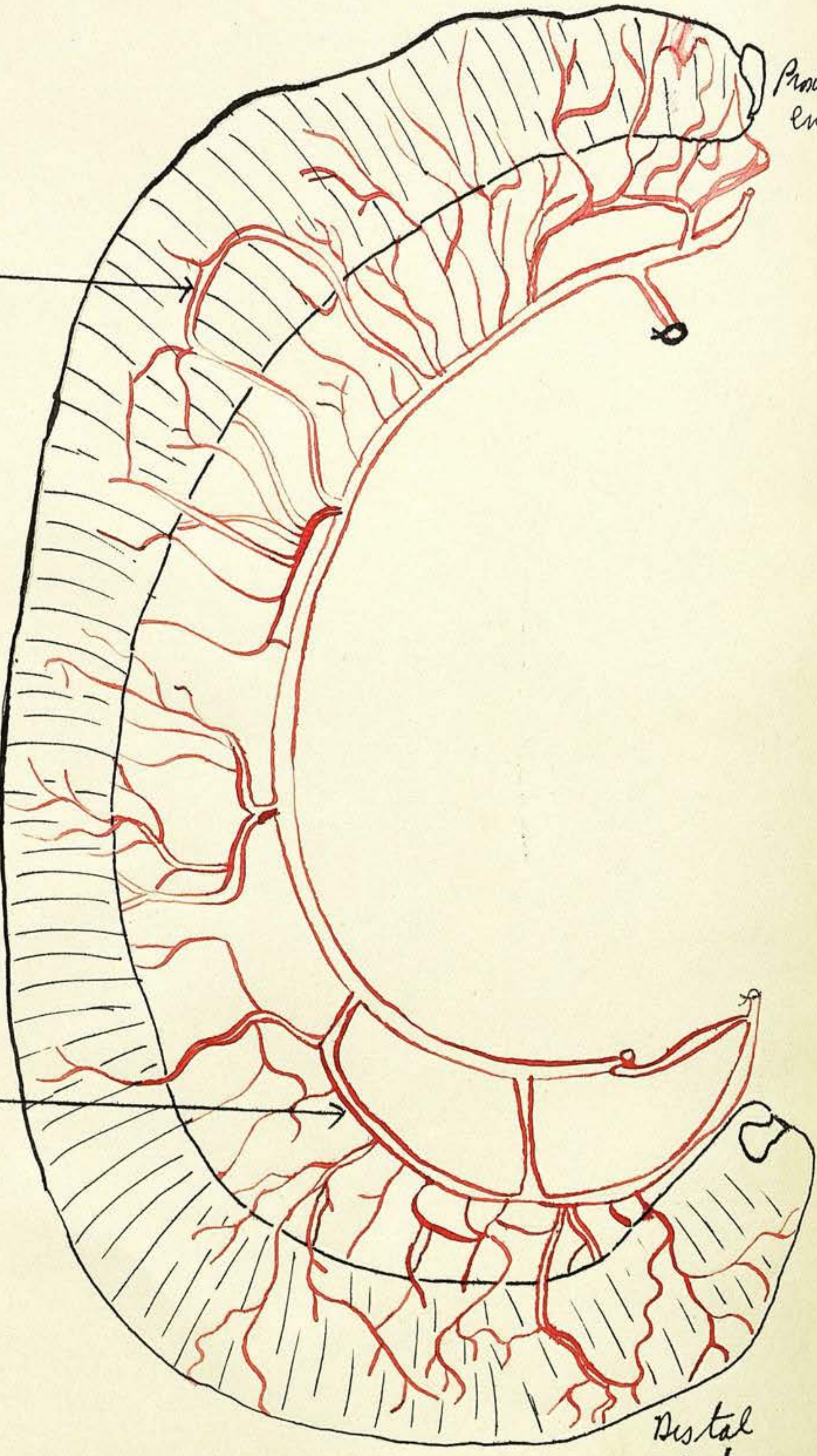
The vasa longa divided into smaller branches which sank into the muscle coat at the taeni coli. No anastomosis was noted from one side of the colon to the other, across the anti-mesocolic border. A secondary arcade formation was developed across the bases of the vasa longa, opposite the bifurcation of primary arcades. An incomplete tertiary arcade system was developed across the vasa longa on the intestinal wall.

Large appendices epiploicae were present marking the course of the vasa longa. Two of these were ligated at their bases; the injection material flowed on into the vessels beyond this ligature.

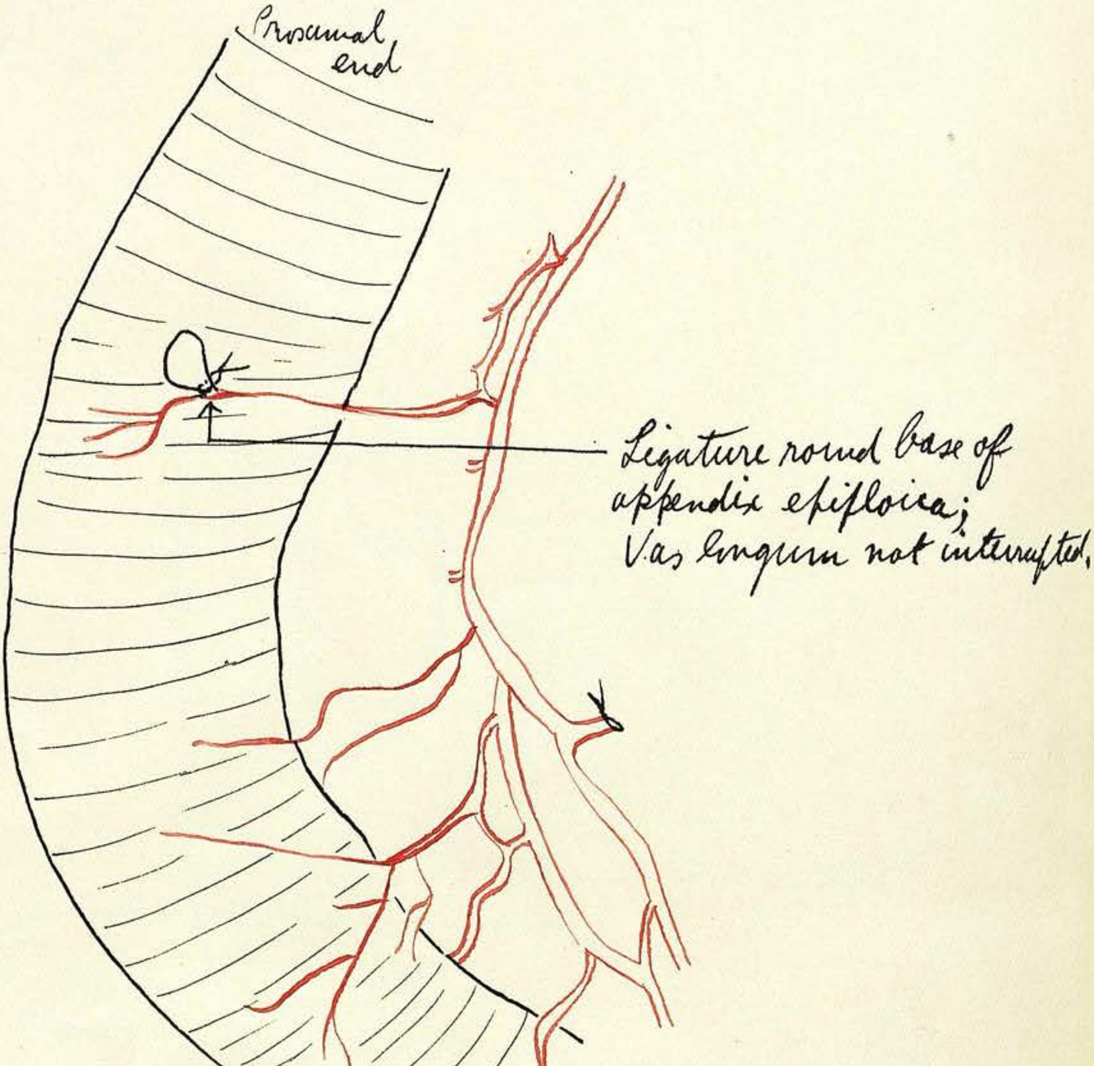
Nos. 16 and 17 (Drawings)/

No 16

Loop of descending iliac and upper pelvic colon viewed from posterior aspect



Loop of iliac colon.



Nos. 16 and 17 (Drawings)

Colon (10) Female aged 24. Died of rheumatic fever.

Specimen - 19 inches of descending iliac and upper pelvic colon. Thin fat free.

A continuous (marginal) artery was present, and a series of arcades between this vessel and the colon wall, progressively larger towards the inferior end of the specimen. Secondary arcades, and well marked tertiary arcades were also present, the tertiary arcades occurring between vasa longa of the same distance apart as those without anastomotic channels present.

The vasa longa arose mostly singly; some came off a short common stem which bifurcated the vessels going to alternate sides of the colon. At one place an anastomosis at the anti-mesocolic border was present between vasa longa from opposite sides. No loop formation was seen in 22 vasa longa counted on the anterior aspect of this specimen.

A regular row of thin appendices epiploicae was present on the anterior aspect of this colon, each marking the course of a vas longum. One was ligated at the base, without preventing the flow of injection material in the course of the vas longum beyond it. No loops were seen in this specimen.

Colon (11) See Appendix.

Colon (12) Male aged 55. Died of pulmonary infarct (old rheumatic history).

A "false" appearance of tortuosity in the course of the vessels was produced in this specimen by the contraction of the specimen on exposure to the stronger percentage of acid.

No loop was detected in the base of the appendices epiploicae. The vasa longa arose

(1) singly

(2) a pair off a common trunk of variable length.

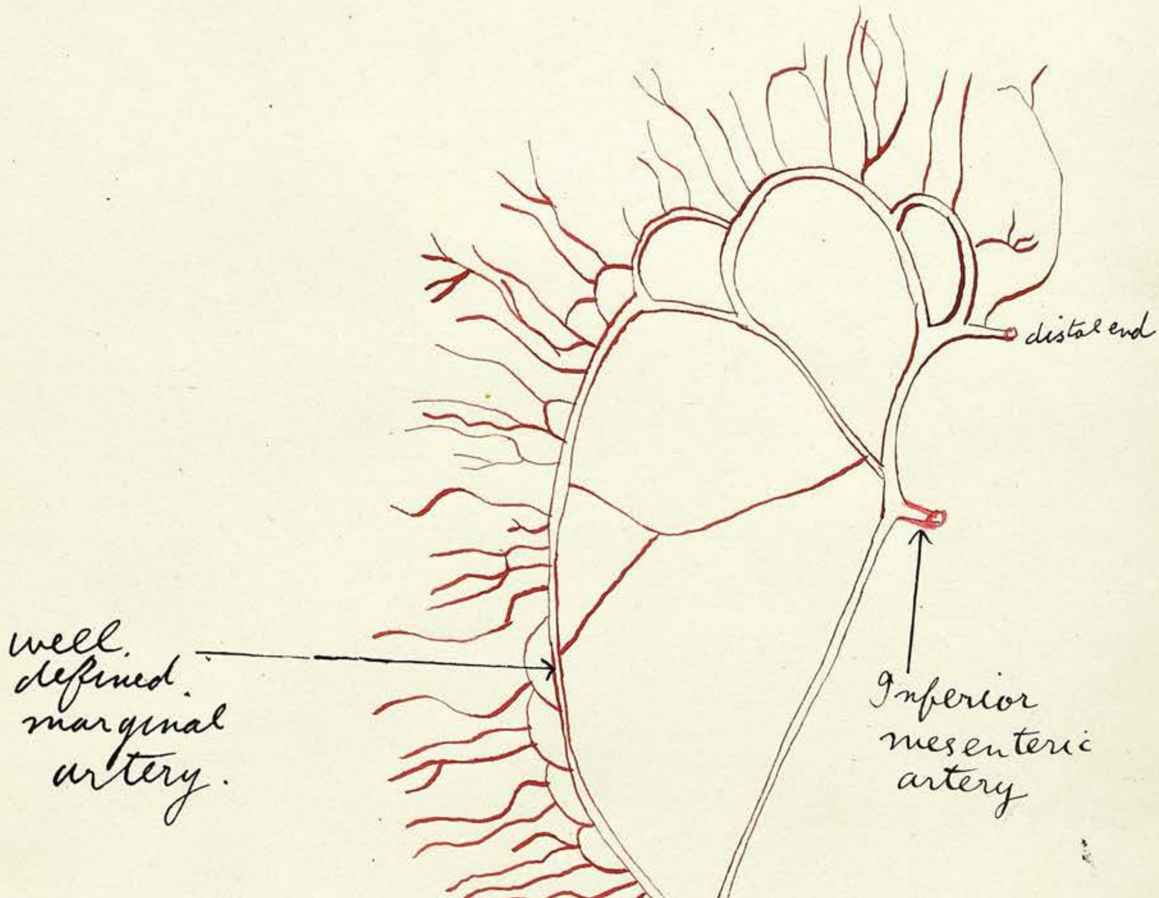
A large number of vessels intermediate in size between vasa longa and brevia were present.

No. 18 (Drawing)/

96

no 18

Vessels of a loop of descending iliac and pelvic colon of a still born infant. Colon wall corroded off.



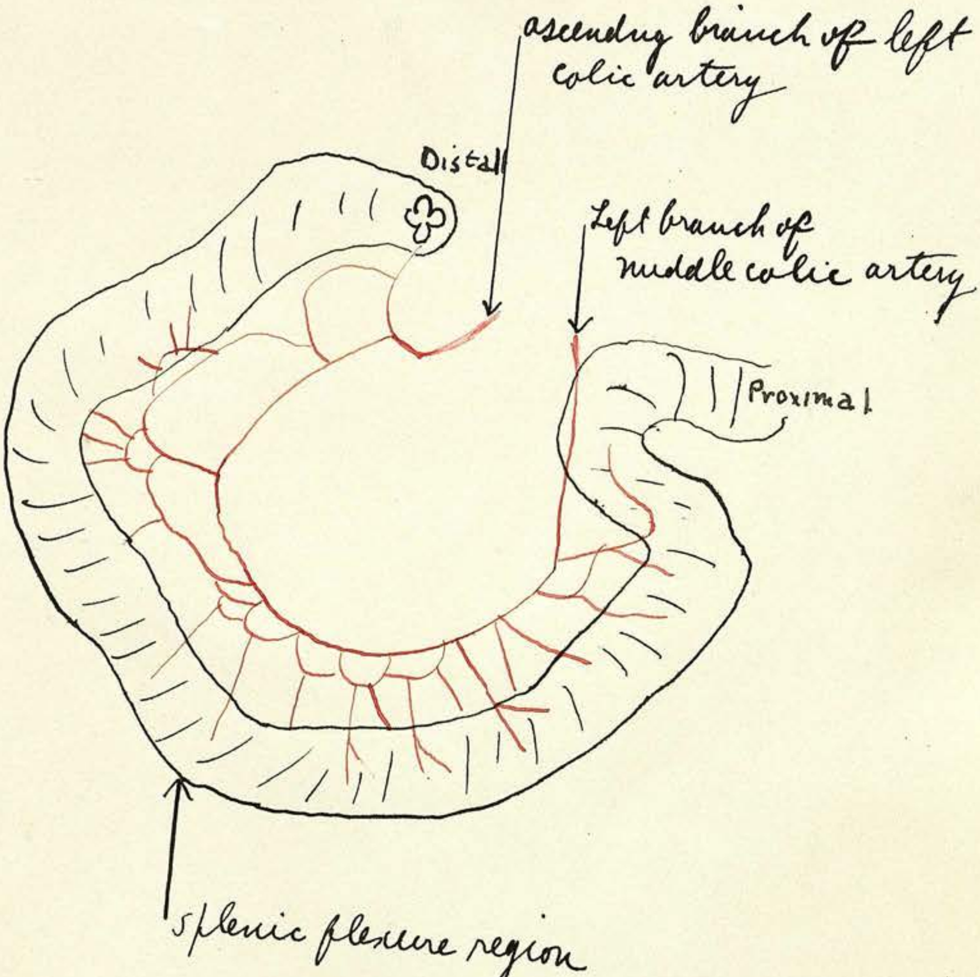
No. 18 (Drawing)

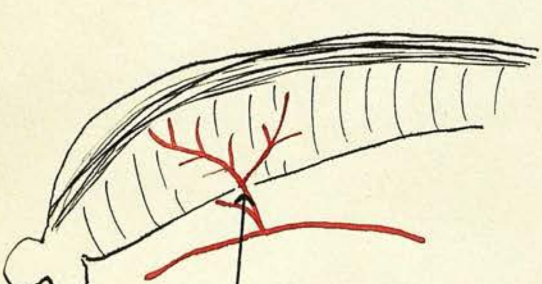
Colon (13) Still-born infant.

Specimen - descending iliac and pelvic colon.

The specimen showed the first, second and third sigmoids with the continuation of the inferior mesenteric as the superior rectal artery. Well defined primary arches and a marginal artery were present.
A series of secondary arcades were also present, and an incomplete development of tertiary arcades.

The appendices epiploicae were thread like processes devoid of fat.
No arterial loop was present in their bases.





Unusual vessel replacing 2 vasa longa.

<u>No. 19 (Drawing)</u>

<u>Colon (14)</u> 7 months foetus.

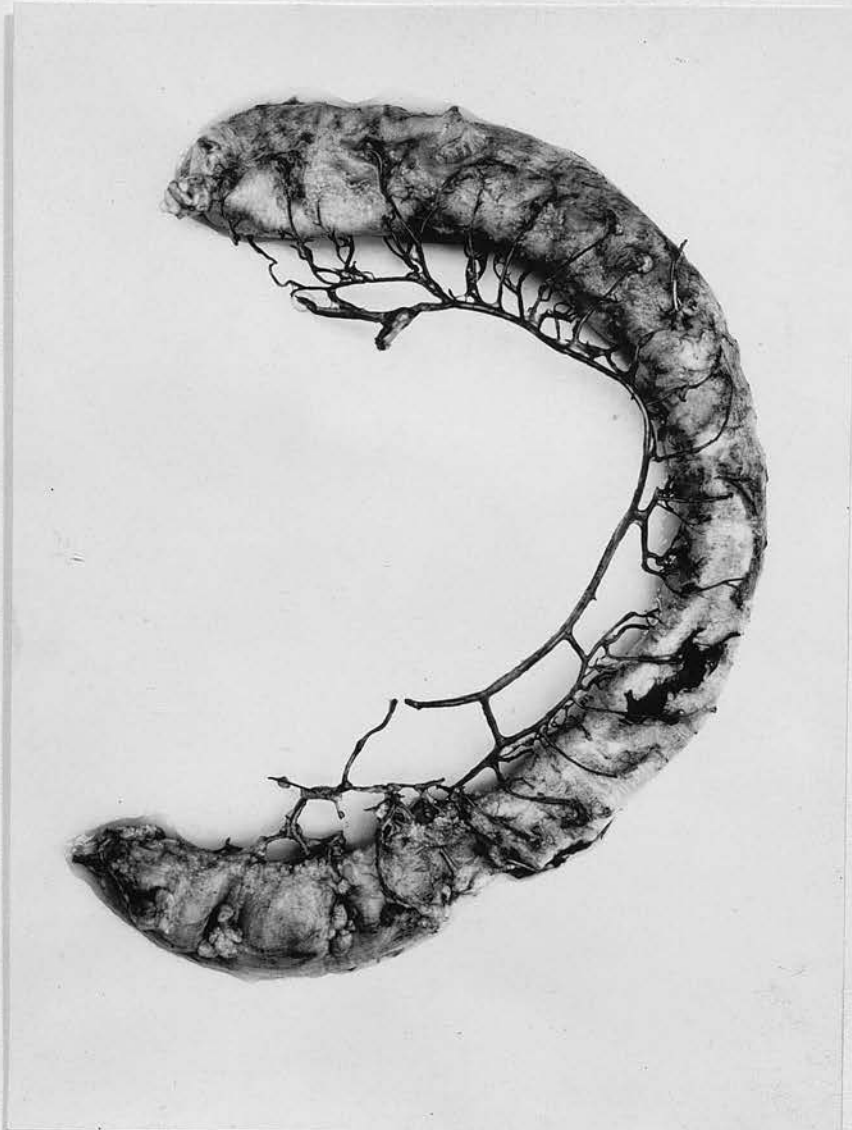
Specimen showed the ascending branch of the left colic, the left branch of the middle colic, and a well defined marginal artery. Primary arcades present, larger towards the inferior end of the specimen. Well developed system of secondary arcades. One unusual vessel noted, replacing two vasa longa, the angle of the bifurcation of the two branches to the same side being practically 90°.

No vasa brevia injected in this specimen, and no appendices epiploicae seen.

No. 20 (Photograph)/

no 20

Descending, iliac and Pelvic colon, partly corroded.



PROXIMAL END

DISTAL END

note marginal artery and origins of vasa longa.

No. 20 (Photograph)

Colon(15) Male aged 14 died of rheumatic fever.

Specimen - descending iliac and pelvic colon.

Photograph shows partly corroded specimen.

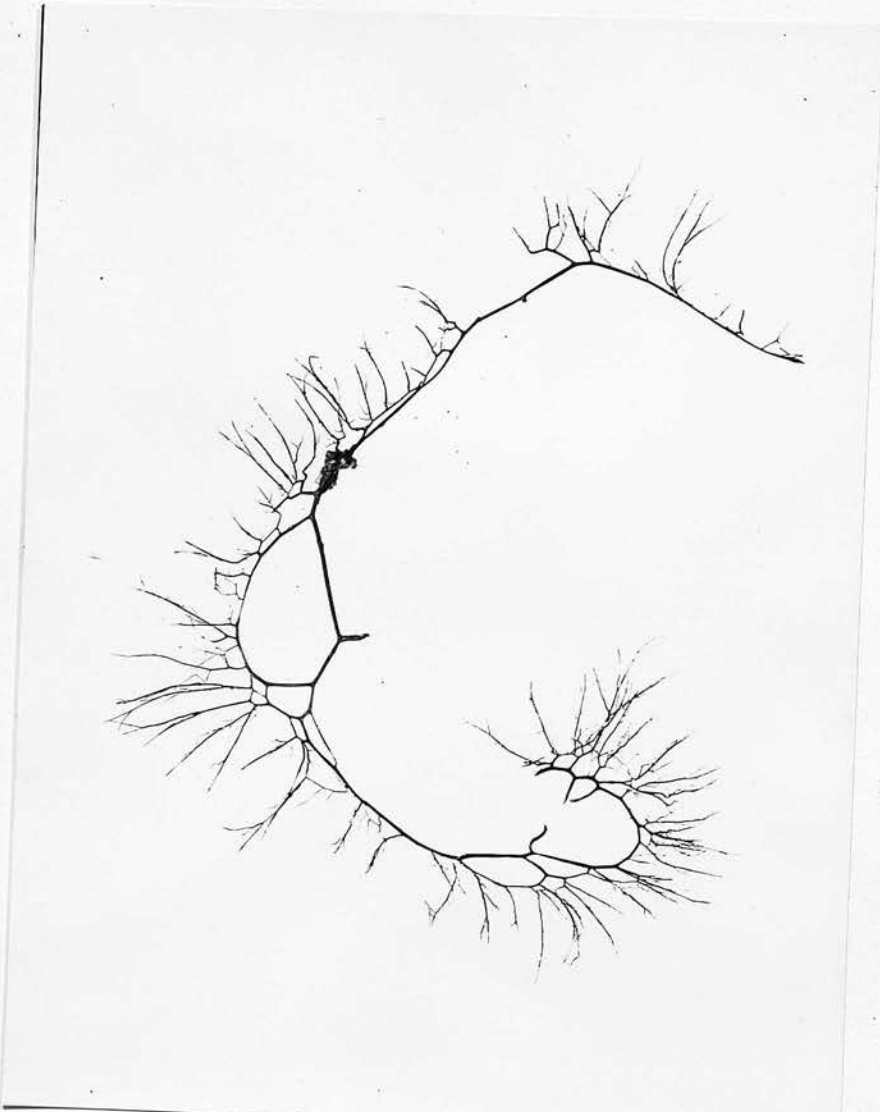
Marginal artery at superior end leads down into arcade formation towards the distal end, (partly broken up). Vasa longa seen coming off to both sides with secondary arcade formation across their bases.

No loop development on the vasa longa seen.

No. 21 (Photograph)/

no 21

Cast of vessels of descending, iliac and pelvic
Colon of a full term foetus. corrosion specimen.



PROXIMAL END

DISTAL END

<u>No. 21 (Photograph)</u>

<u>Colon (15a)</u> Full term still-born foetus.

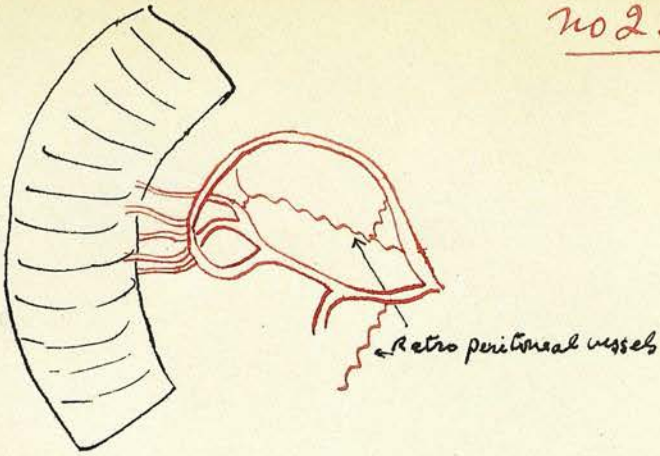
Specimen - descending, iliac and pelvic colon.

Photograph shows the marginal artery, with anastomotic channels across the bifurcation of the feeding vessels, a frequent secondary arcade formation across the proximal ends of the vasa longa, and the vasa longa continuing with vasa brevia branching from them.

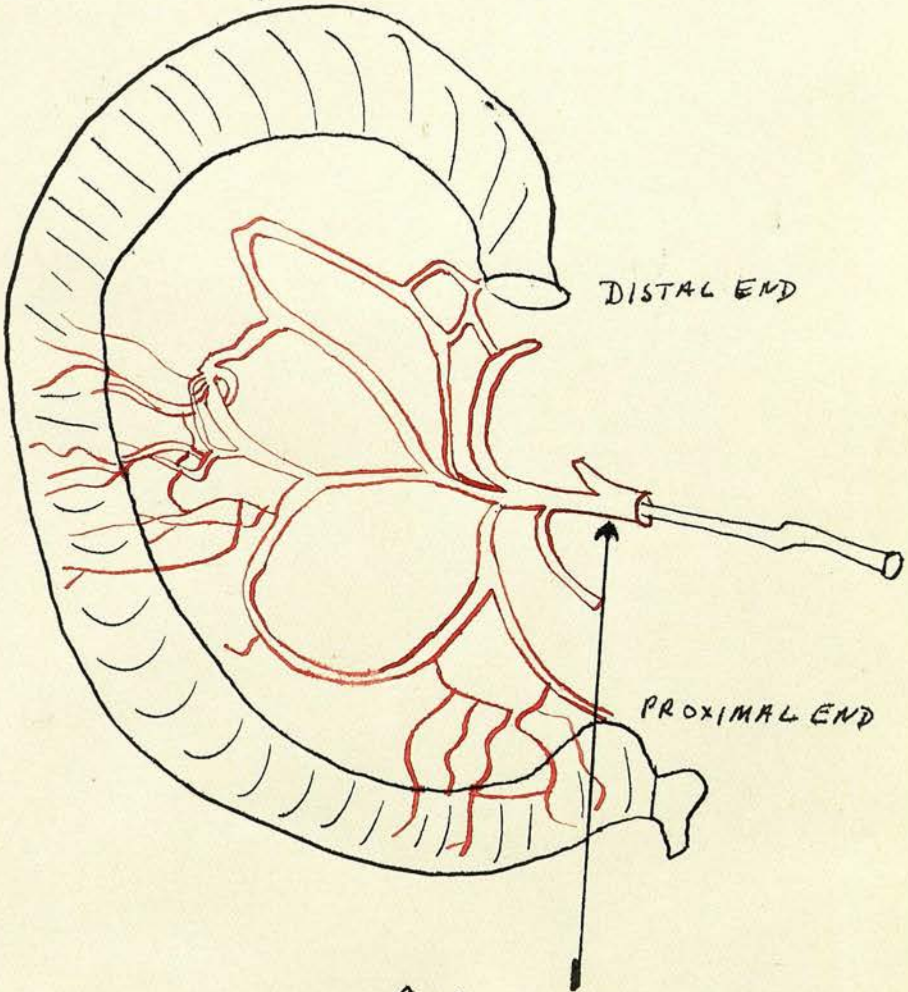
No anastomosis between vasa longa at anti-mesocolic border.

No. 22 (Drawing)/

Segment (of colon drawn below) showing the slender retroperitoneal vessels, branches of the intestinal vessels.



Descending, iliac and pelvic colon of full term foetus.



No. 22 (Drawing)

Colon (16) Full term still-born foetus.

Specimen - descending, iliac and pelvic colon.

Note arcades across bifurcation of main vessels, formed by slender vessels, one on the colon wall.

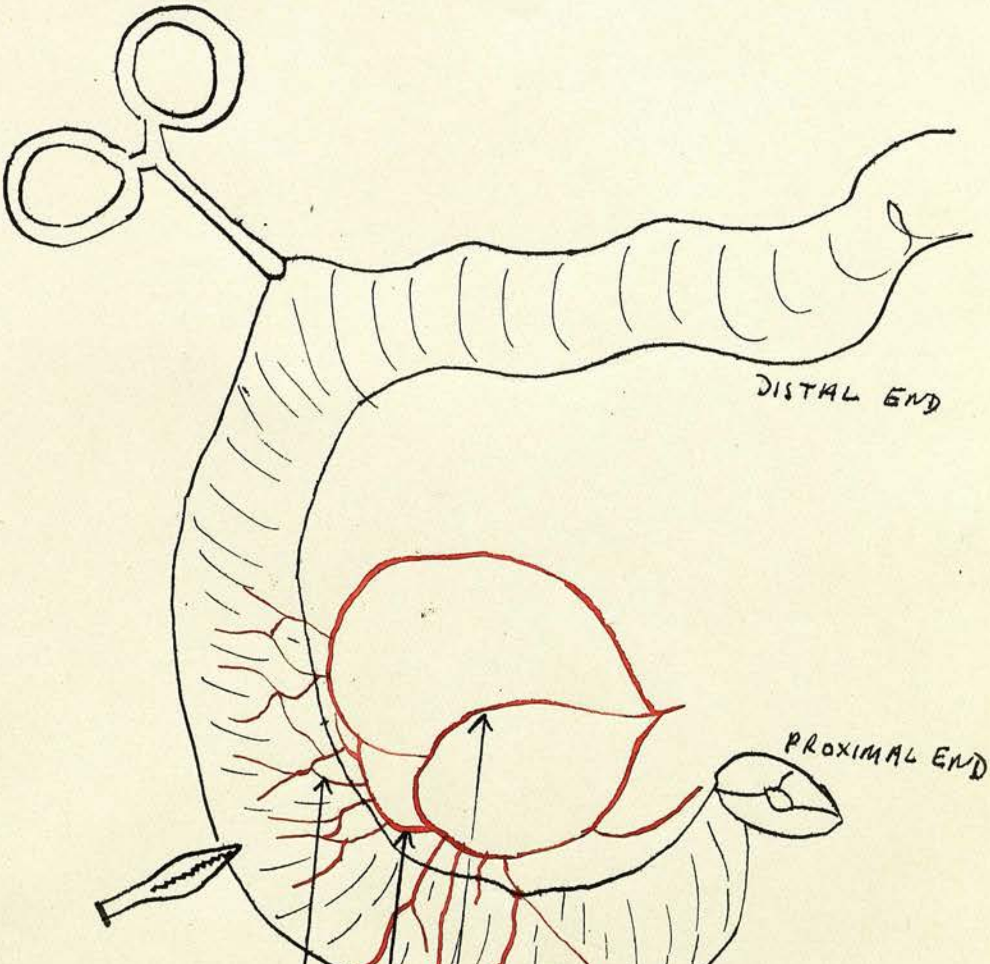
Note also the slender retroperitoneal vessels which have become injected.

After partial corrosion in 50% HCl for 48 hours, the muscular coat was stripped off. The terminations of the vasa longa were seen, with several partially injected channels connecting the vasa longa from opposite sides of the colon, at the anti mesocolic border.

Nos. 23 and 24 (Drawings)/

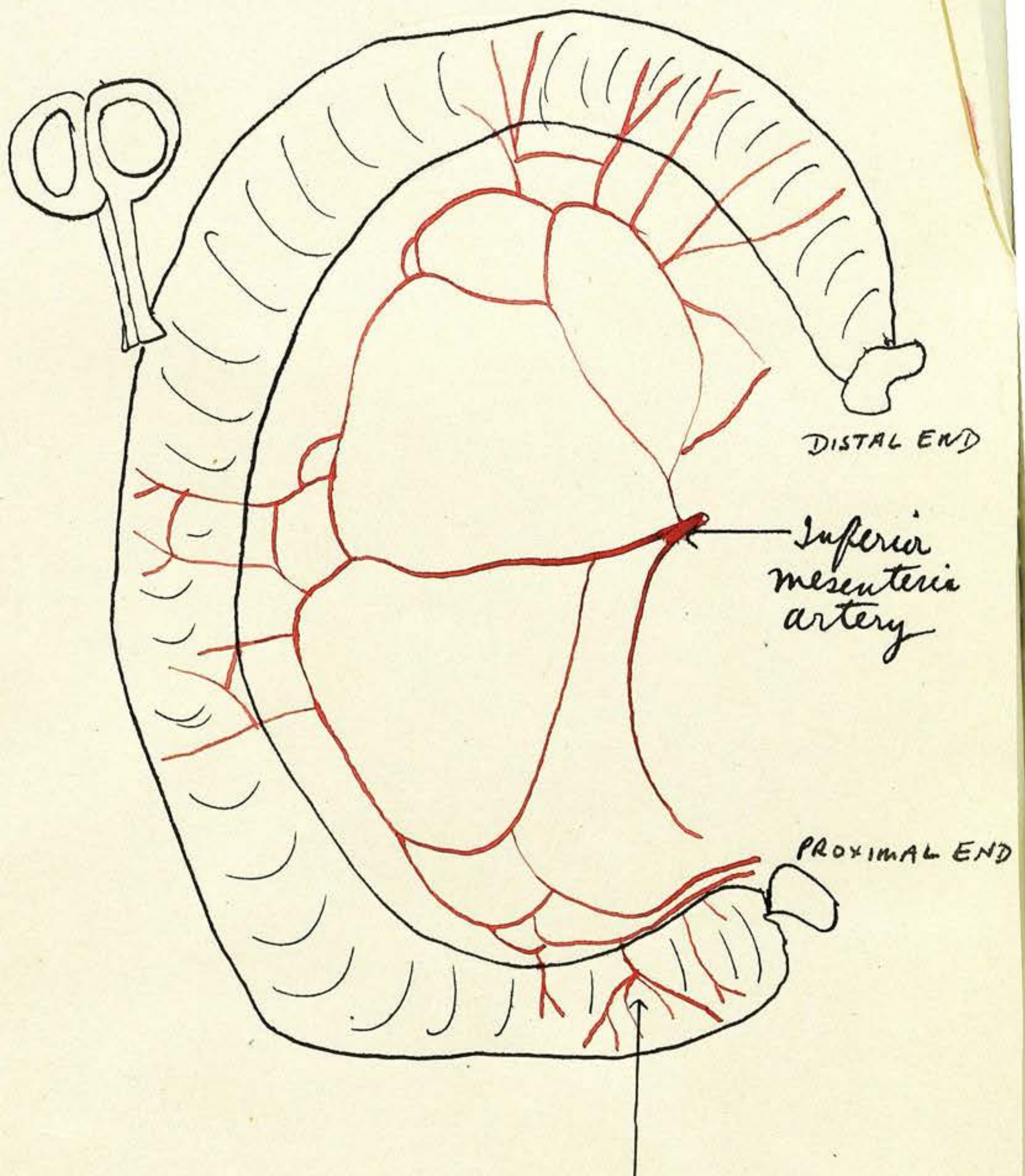
no 23

Descending, iliac and pelvic colon of infant 5 months old.



Showing details of :- Tertiary, Secondary, Primary } arterial arcades.

Descending, iliac, and pelvic colon of infant 5 months old. (same as 23)



Unusual vessel, forking to supply an area equal to three vasa longa.

Nos. 23 and 24 (Drawings)

Colon (17) Infant 5 months old died of icterus neonatorum.

Specimen - descending, iliac and pelvic colon.

Inferior mesenteric artery injected.

Primary arcades produced by sigmoid arteries present. Vasa longa and brevia present, the vasa longa rising

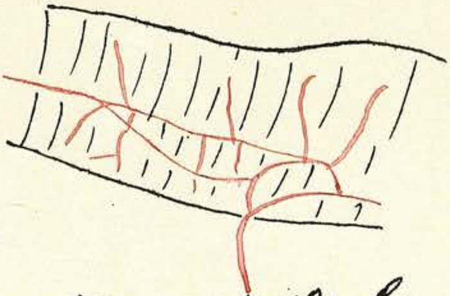
(1) singly

(2) by a common trunk for two

(3) one big vessel forking to supply an area equal to three vasa longa.

Tertiary arcades noted on **posterior** aspect of colon. Appendices epiploicae present, tiny pellets in line with the vasa longa.

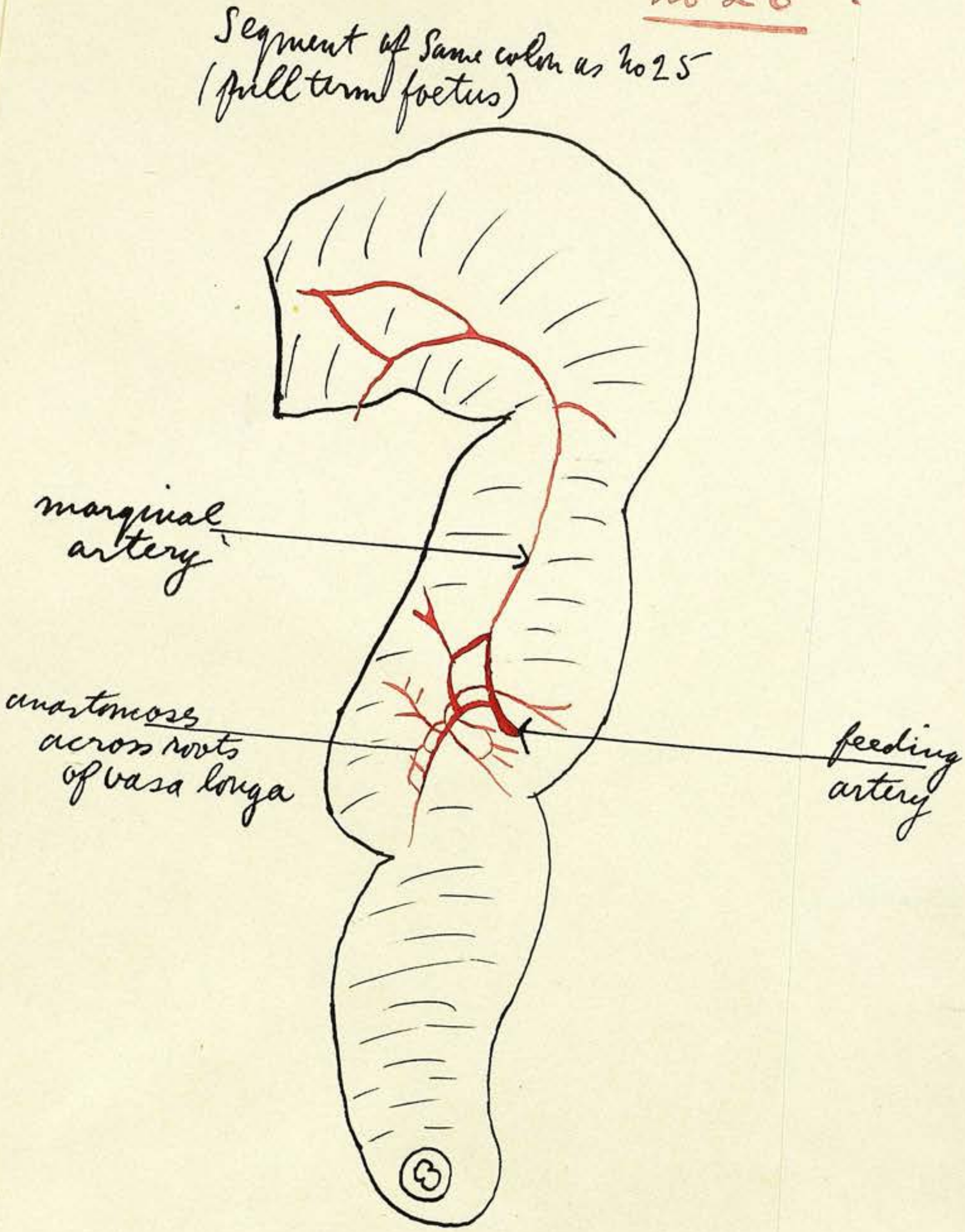
Nos. 25 and 26 (Drawings)/



Segment of colon showing detail of origin of vasa longa.

Segment of colon showing arcades produced by anastomoses across bifurcations

no 26


Segment of Same colon as no 25
(full term foetus)
marginal artery
anastomoses across roots of vasa longa
feeding artery

Nos. 25 and 26 (Drawings)

Colon (18) Still-born foetus.

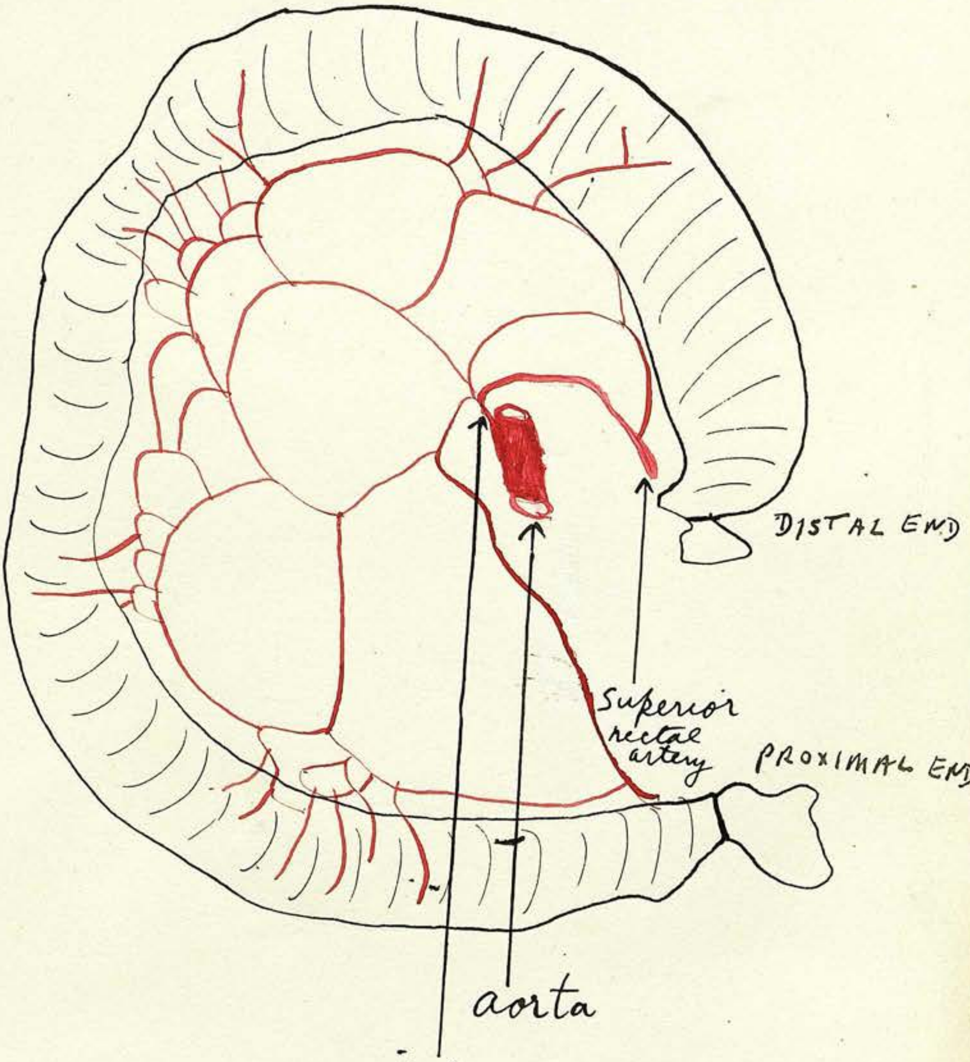
Inferior mesenteric artery injected.
Gross leakage.

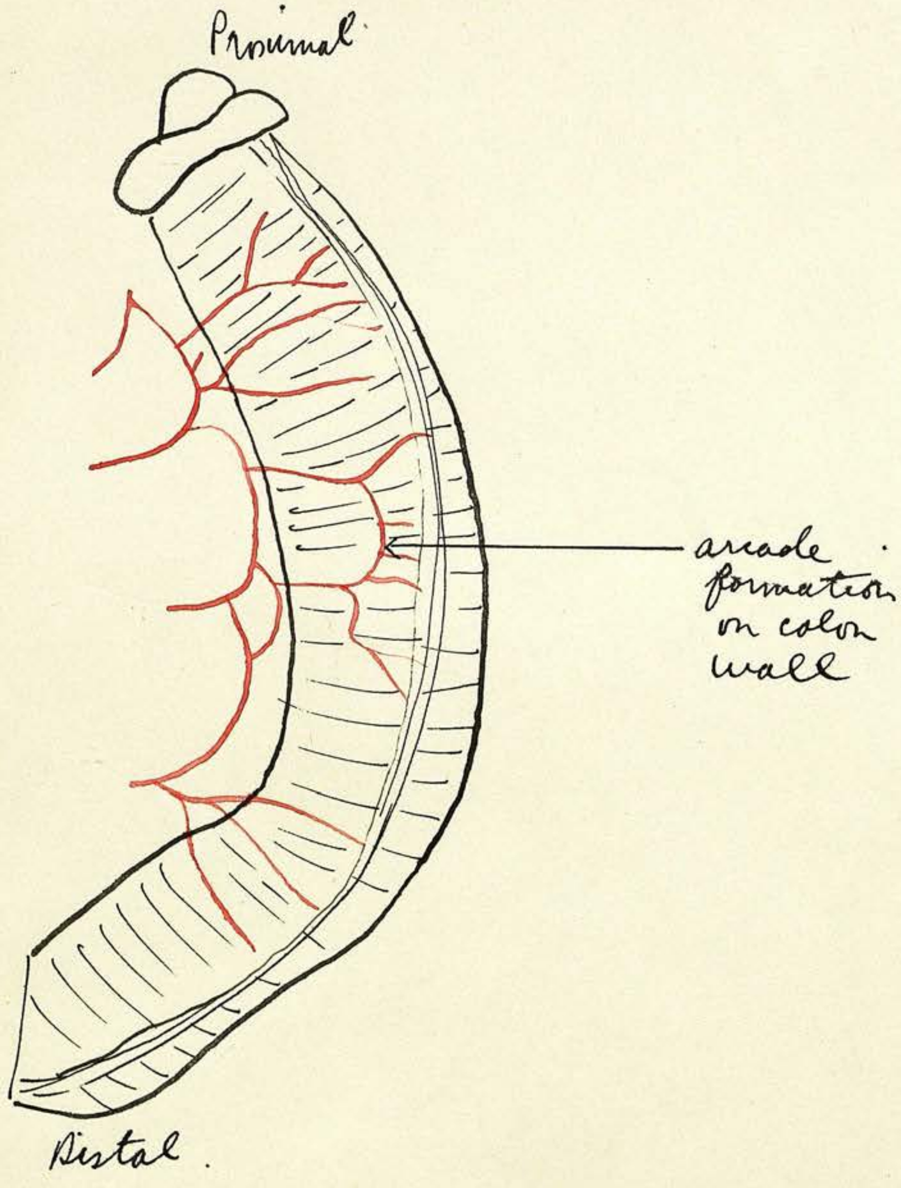
Specimen showed fairly regular alternate origin of the vasa longa; also showed increasing development of secondary arcades at the distal end of the specimen.

Nos. 27 and 28 (Drawings)/

no 27

Descending, iliac and pelvic colon of full term foetus.




Proximal
arcade formation on colon wall
Distal.

Nos. 27 and 28 (Drawings)

Colon (19) Full term female foetus.

Specimen - descending, iliac and pelvic colon.

Aorta injected with 6% celloidin stained alkanin. Leaked after half an hour.

Note well developed primary arcades, and numerous secondary arcades across bifurcations, and origins of vasa longa.

Occasional tertiary arcade present.

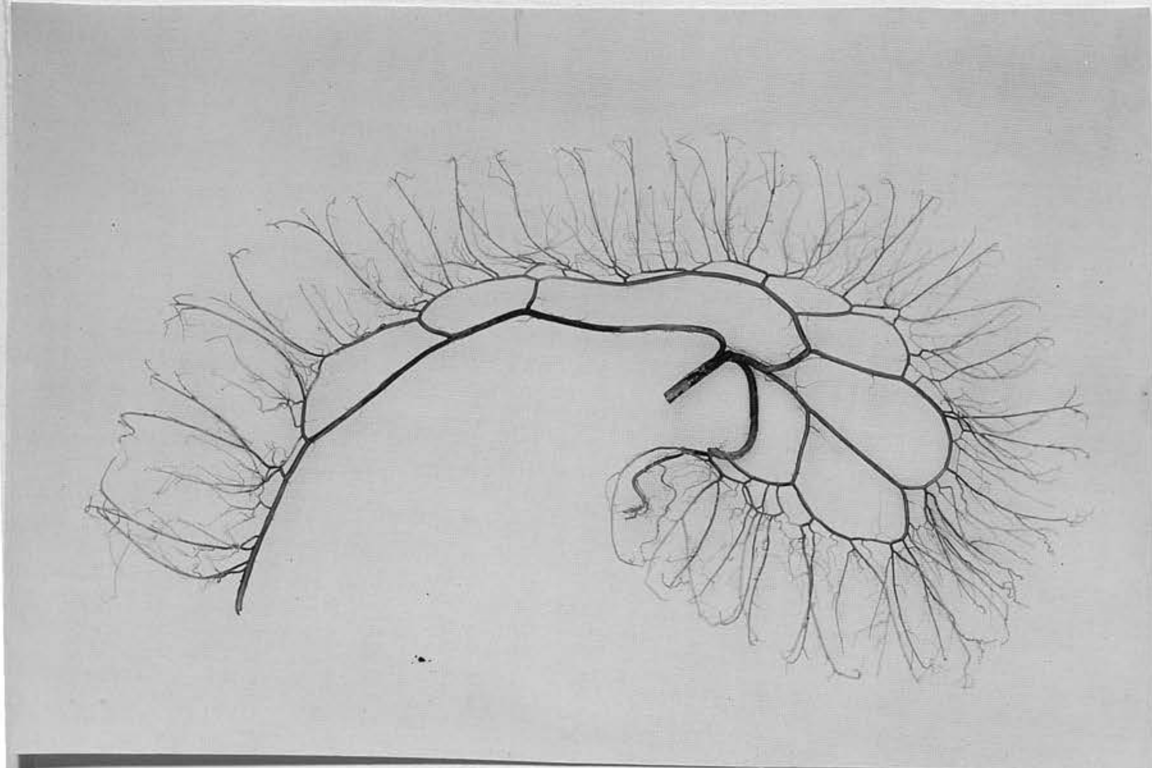
The appendices epiploicae were tiny pellets of fat on the course of the vasa longa.

No arterial looping was seen anywhere.

Colon (20) See Appendix.

Nos. 29 and 30 (Photographs)

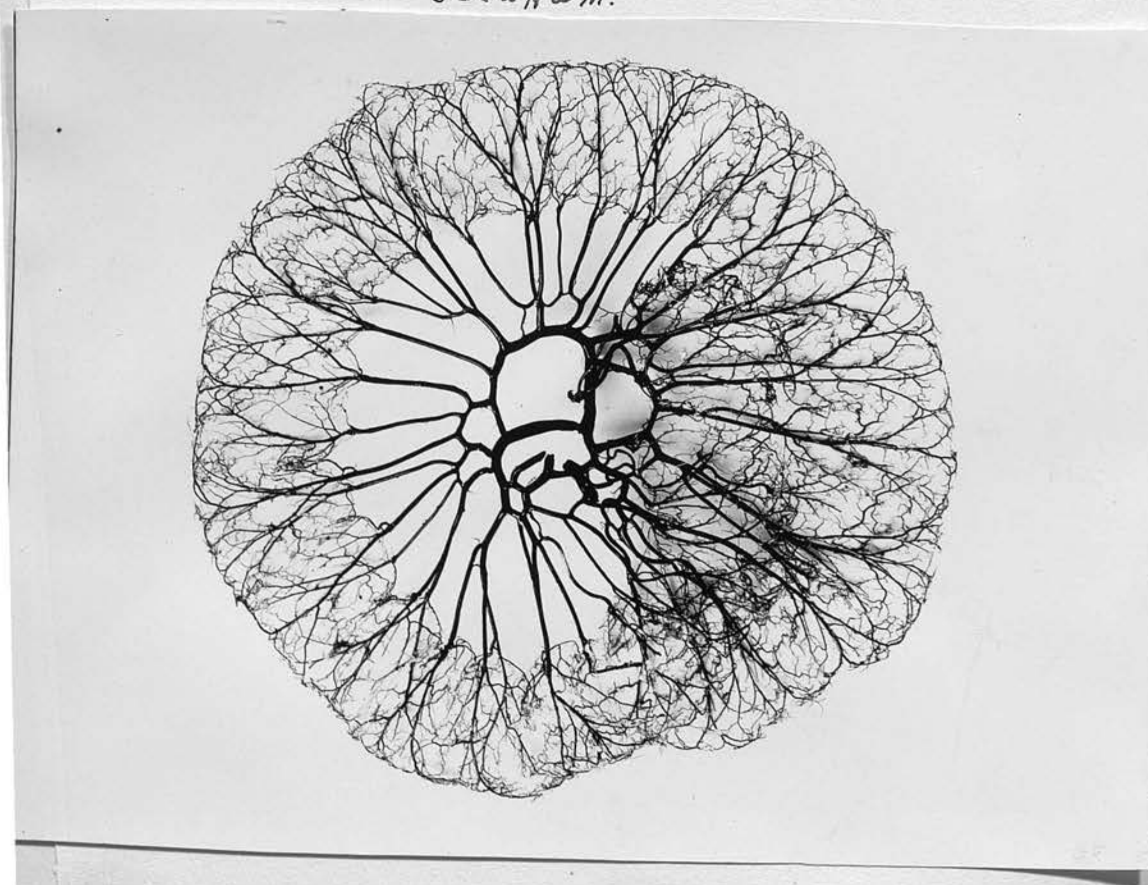
Corrosion cast of descending, iliac and pelvic COLON's arterial pattern (adult) no. 29, and no. 30



Inferior mesenteric artery injected. marginal artery, and arcade formation in pelvic mesocolon demonstrated also vasa longa & brevia seen

DISTAL. END

JEJUNUM.



Corrosion cast of vessels of jejunum from same cadaver inserted here as a contrast to the vessels of the colon. note much greater vascularity, and free antimesenteric anastomoses.

Nos. 29 and 30 (Photographs)

Colon (21) Male aged 40. Died fractured skull and extradural haemorrhage.

Specimen - descending and pelvic colon.

Corrosion cast photographed. This shows the marginal artery at the proximal end, and the arcade formation at the distal end.

The secondary arcades across the origins of the vasa longa are also well seen, and the regularity of the size of the vasa longa. In one or two places there is an anastomosis between the vasa longa on the anti mesocolic border.

In contrast with this, the photograph of a loop of upper jejunum of the same case is shown (fully described later). The vastly richer blood supply of the jejunum, with frequent anastomosis along the anti mesenteric border is well seen in this photograph.

Colon (22) Still-born foetus.

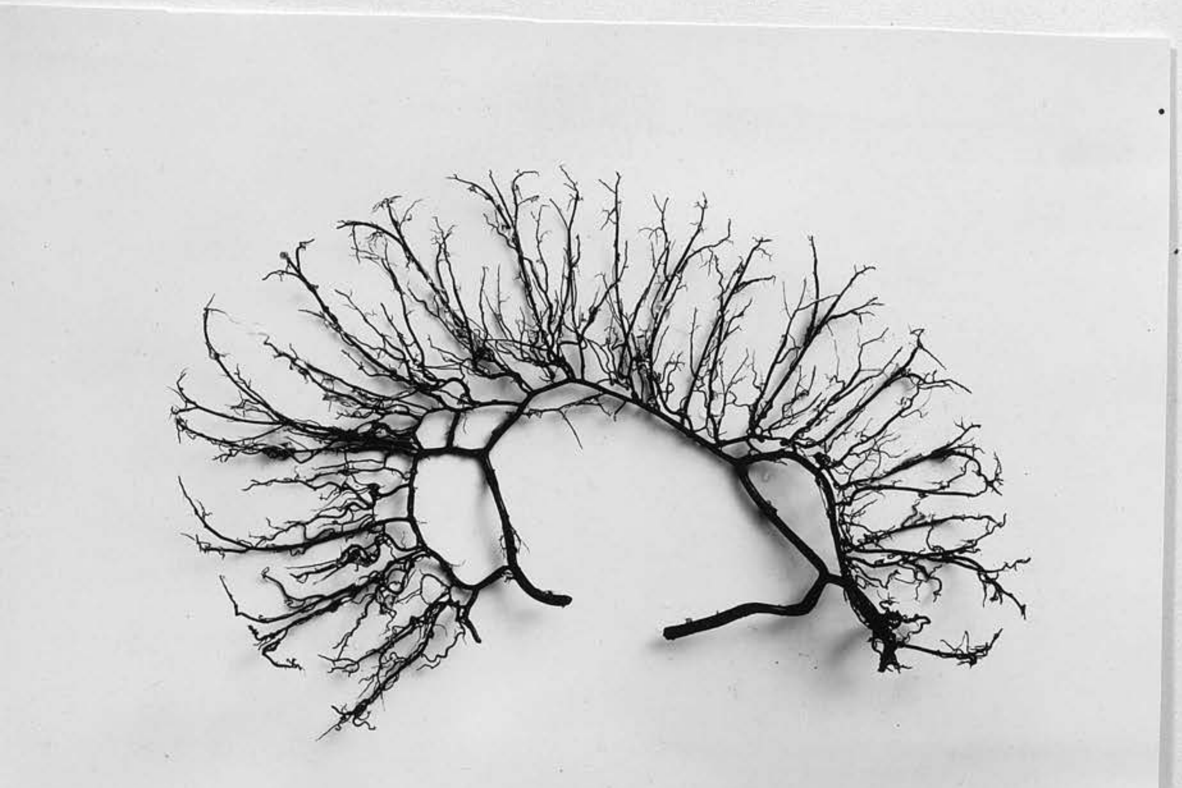
Specimen - descending, iliac and upper pelvic colon.

Inferior mesenteric artery injected. Marginal artery proximally, primary arcades distally noted.

Vasa longa seen ramifying with their fellows of the opposite side in several places. Well developed tertiary arcade system present in this specimen on the gut wall.

No 31

Corrosion cast of arteries of Descending, iliac and pelvic colon of a boy aged 5.



DISTAL END. PROXIMAL END

Inferior mesenteric artery

No. 31 (Photograph)

Colon (23)

Boy aged 5. Died of cerebral haemorrhage.

Specimen - descending, iliac and pelvic colon.

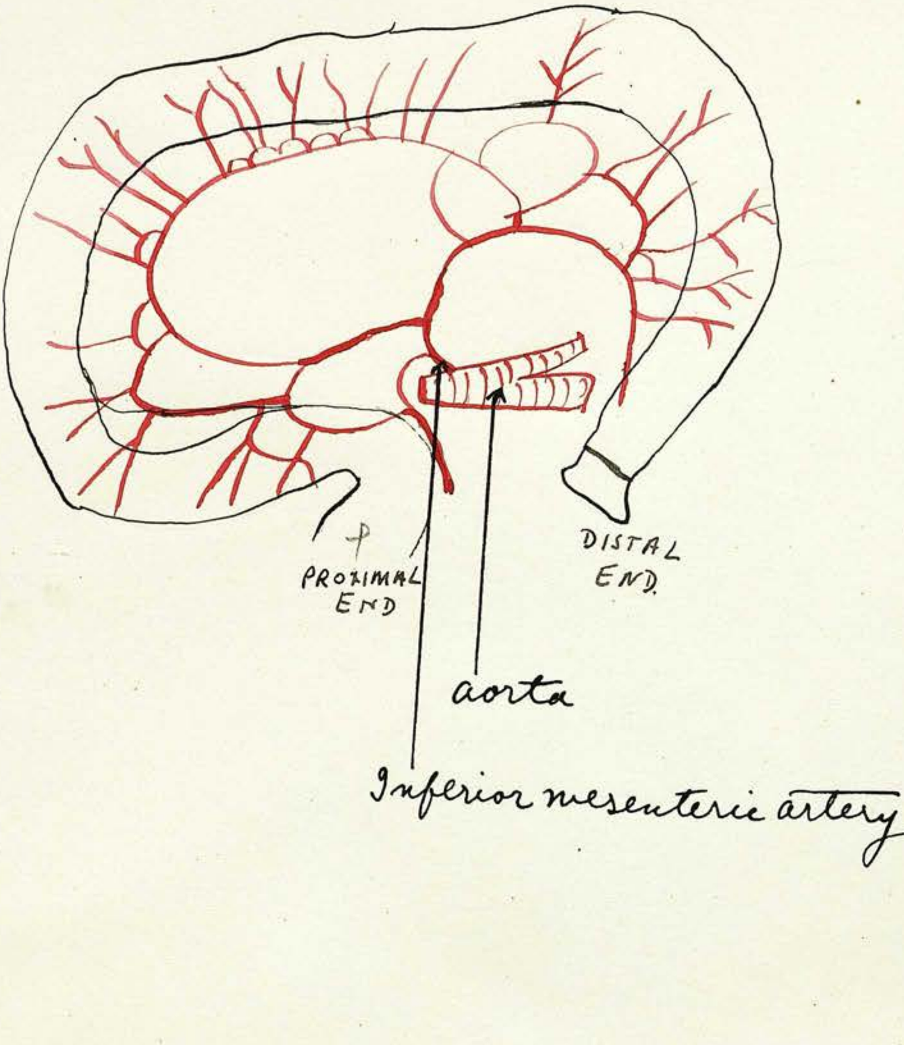
Photograph shows primary and secondary arcades, vasa longa with no anastomosis across anti mesocolic border, numerous vasa brevia and vasa intermedia.

No loop formation.

No. 32 (Drawing)/

no 32

Splenic flexure, descending iliac and pelvic colon
of 7 month foetus.



No. 32 (Drawing)

Colon (24)

Seven months foetus.

Specimen - complete colon.

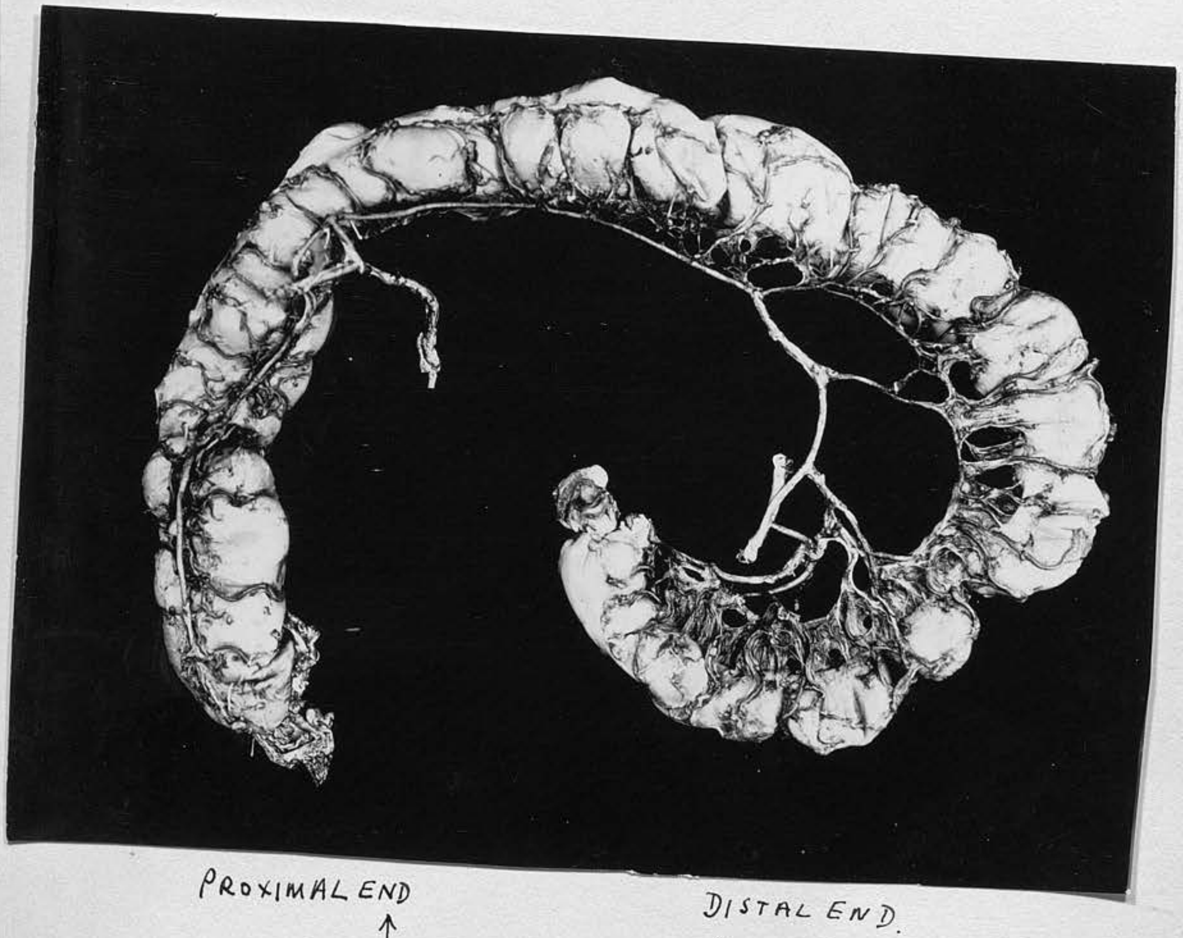
Note primary and secondary arcades, and straight course of vessels.

No development of appendices epiploicae in this specimen.

No. 33 (Photograph)/

No 33

Partially corroded specimen of adult descending, iliac and pelvic colon.



PROXIMAL END

↑

Left colic artery

DISTAL END.

↑

Inferior mesenteric artery becoming superior rectal.

No. 33 (Photograph)

Colon (25).

Specimen - Adult descending, iliac
and pelvic colon.

Photographed.

Note (1) marginal artery

(2) arcade formation by sigmoid arteries, with secondary arcades across bifurcations between roots of vasa longa.

No. (34) Drawing/

No. 33 (Photograph)

Colon (25).

Specimen - Adult descending, iliac
and pelvic colon.

Photographed.

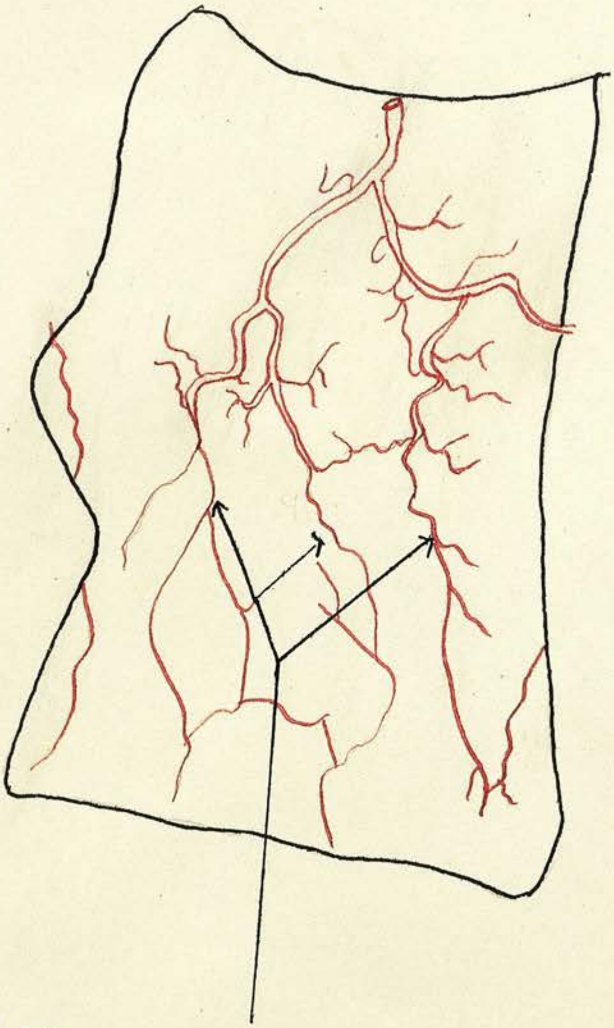
Note (1) marginal artery

(2) arcade formation by sigmoid
arteries, with secondary
arcades across bifurcations
between roots of vasa longa.

No. (34) Drawing/

Portion of wall of adult pelvic colon laid open to show antimesocolic area.

No: 34



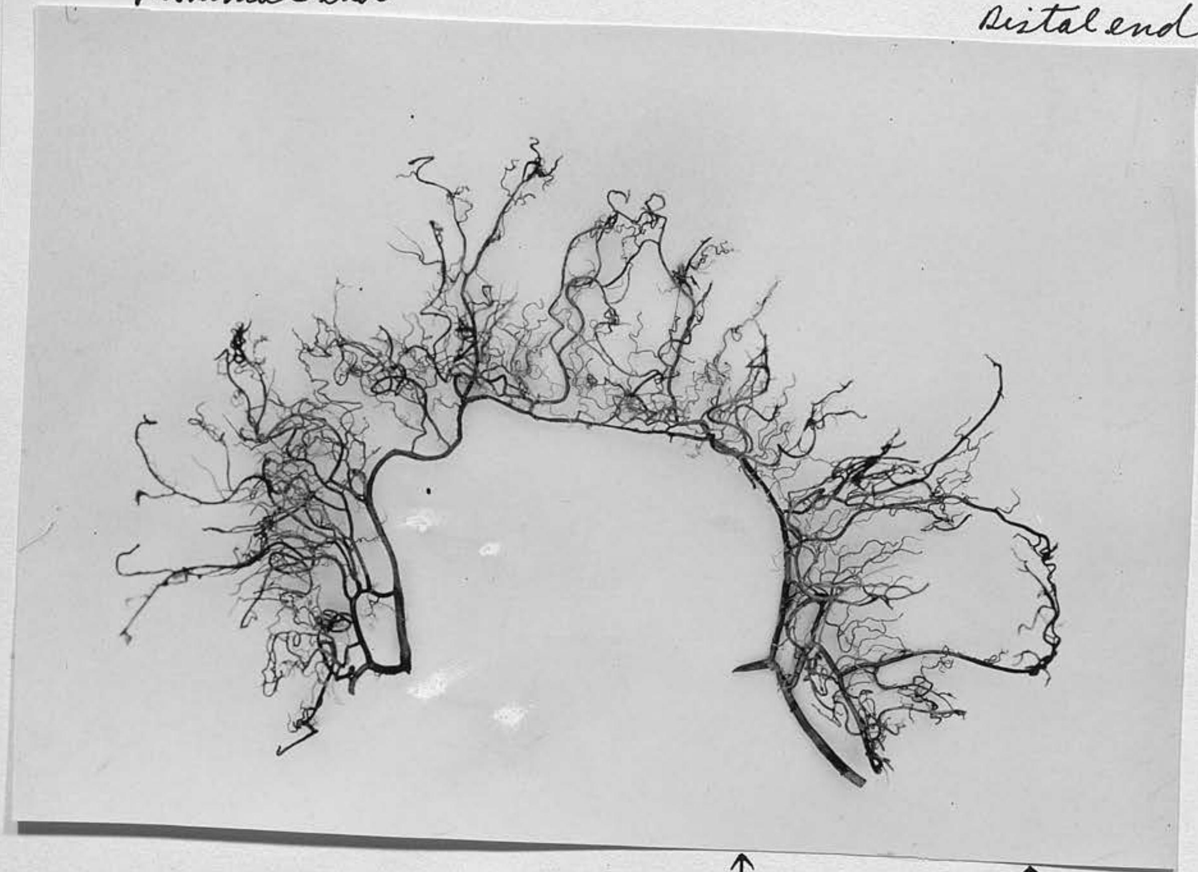
3 vessels anastomosing across the anti mesocolic border.

No 35

Corrosion cast of vessels of adult pelvic colon

Proximal end

Distal end



upper sigmoid artery

These vessels are separate, and do not anastomose

No. 34 (Drawing)

and

No. 35 (Photograph)

Colon (26).

Specimen - Portion of adult pelvic colon.
Corrosion cast.

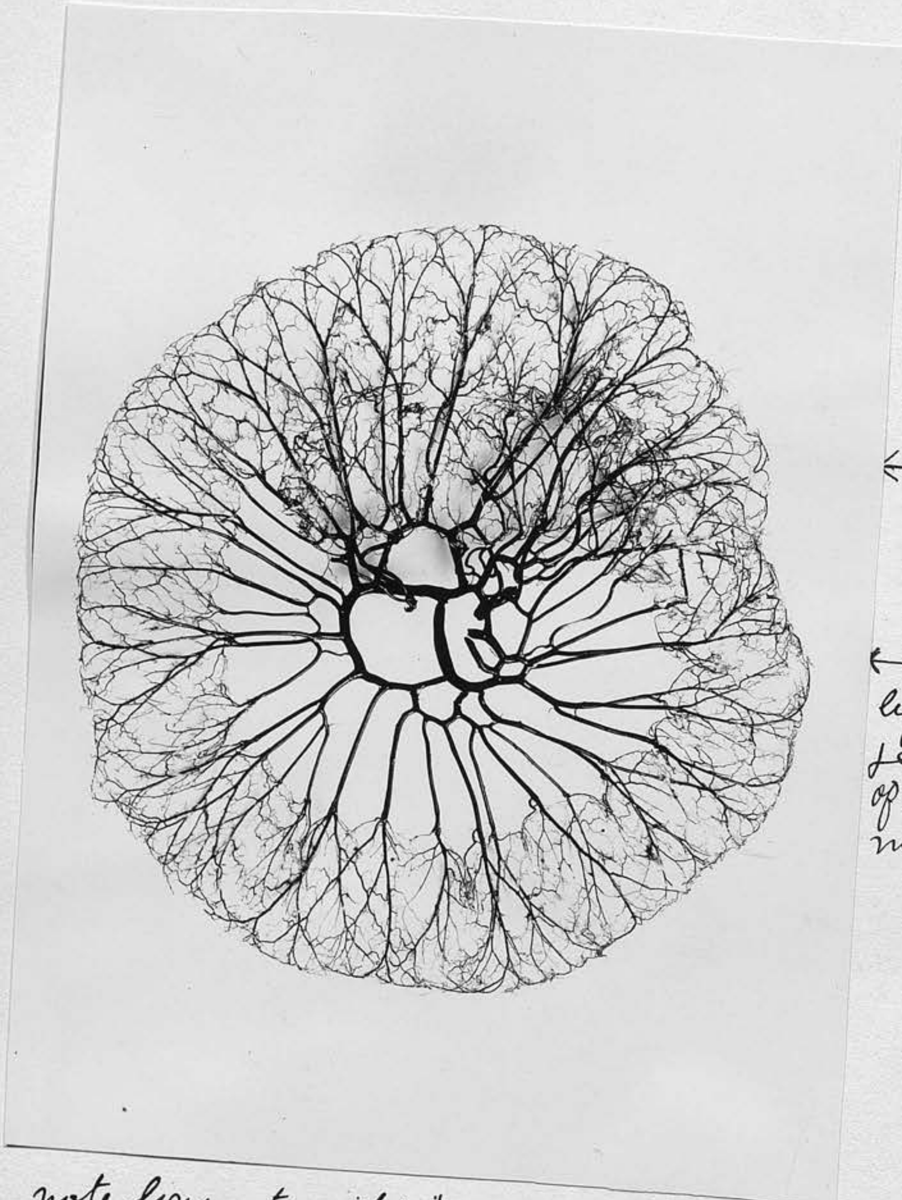
Portion of wall laid open to show anti mesocolic area, where three vessels were seen joining across mid line.

Rest of specimen photographed. Shows marginal artery, anastomoses across roots of vasa longa, Shows also vasa longa and a number of intermediate vessels.

No anastomosis across the mid line visible on the corrosion specimen (vessels at right end of specimen are separate).

Small Intestine Experiments/

Corrosion cast of vessels of adult upper jejunum (same as no. 30).



Upper and lower ends overlapping here.

Injected by a large jejunal branch of the superior mesenteric artery.

note long straight "vasa recta" running without anastomoses till the position of the intestine is reached. note also extremely free anastomoses between the vessels on the gut wall (corroded, but position shown by this vascular net)

SMALL INTESTINE EXPERIMENTS

No. 36 (Photograph)

No. 1. Male aged 40. Died of fractured skull and extra dural haemorrhage.

Specimen - a loop of upper jejunum.

Photograph of the cast shows the loop of upper jejunum in a circle, upper and lower ends overlapping for several inches. Note the long straight vasa recta running without anastomoses till the intestine is reached.

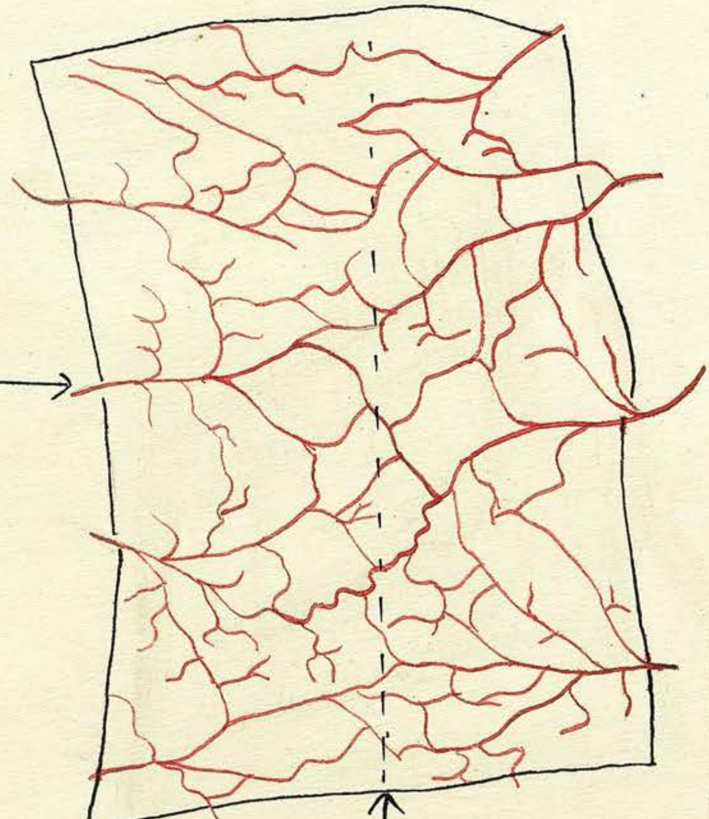
Note the very free anastomoses on the intestine produced by the forking of the vasa recta, making a web of fine vessels completely surrounding the intestine.

There are anastomoses at the anti mesenteric border between vessels of the opposite sides all along this specimen.

No. 37 (Drawing)/

No 37

Specimen of adult small intestine opened out. Partly dissected, the vessels being traced through the coats of the intestine



a vas rectum

Antimesenteric border with free anastomoses across it

<u>No. 37 (Drawing)</u>

and

<u>No. 38 (Photograph)</u>

<u>No. (2)</u>.

Specimen of intestine, jejuno-ileal region.

A portion removed, opened and teased out to show the anastomoses across the anti mesenteric border. 13 anastomotic channels were seen crossing between four vasa recta on each side. These junctions were either

(a) direct

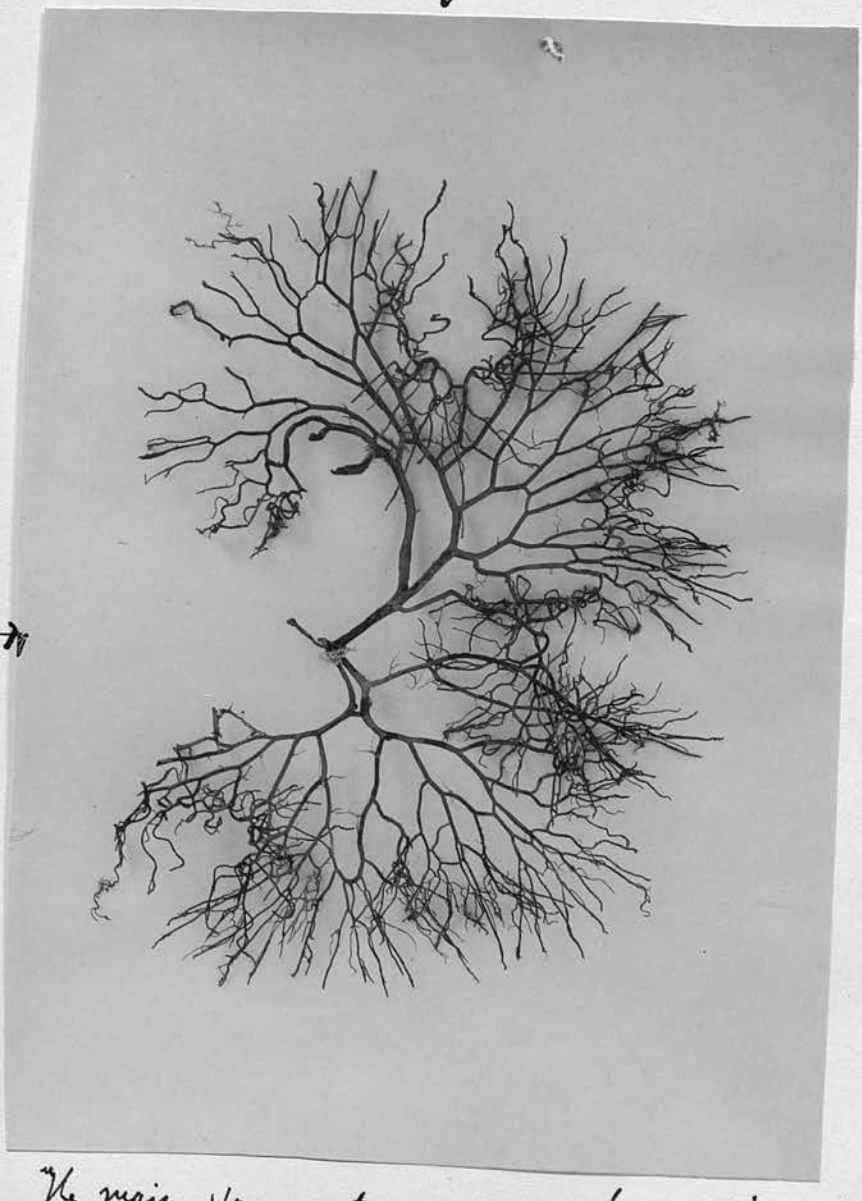
or (b) by a plexiform pattern between arcades.

The remainder of the specimen was then replaced in 50% HCl for a further 6 days, then washed with water.

The cast obtained was photographed. It shows an arcade formation in the mesenteric vessels with vasa recta coming off them. Anastomosis between vasa recta of opposite sides occurs all along this specimen, and can be seen in the photograph in several places.

no 39

Corrosion cast of adult lower jejunum.
Injected via a jejunal branch of the superior mesenteric artery.



jejunal artery injected

The main vasa recta are seen; the injection material has not flowed up into the finest ramifications. Note the constant anastomosing channel across bifurcating vessels.

No. 39 (Photograph)

No. (3), Adult.

Specimen - lower jejunum.

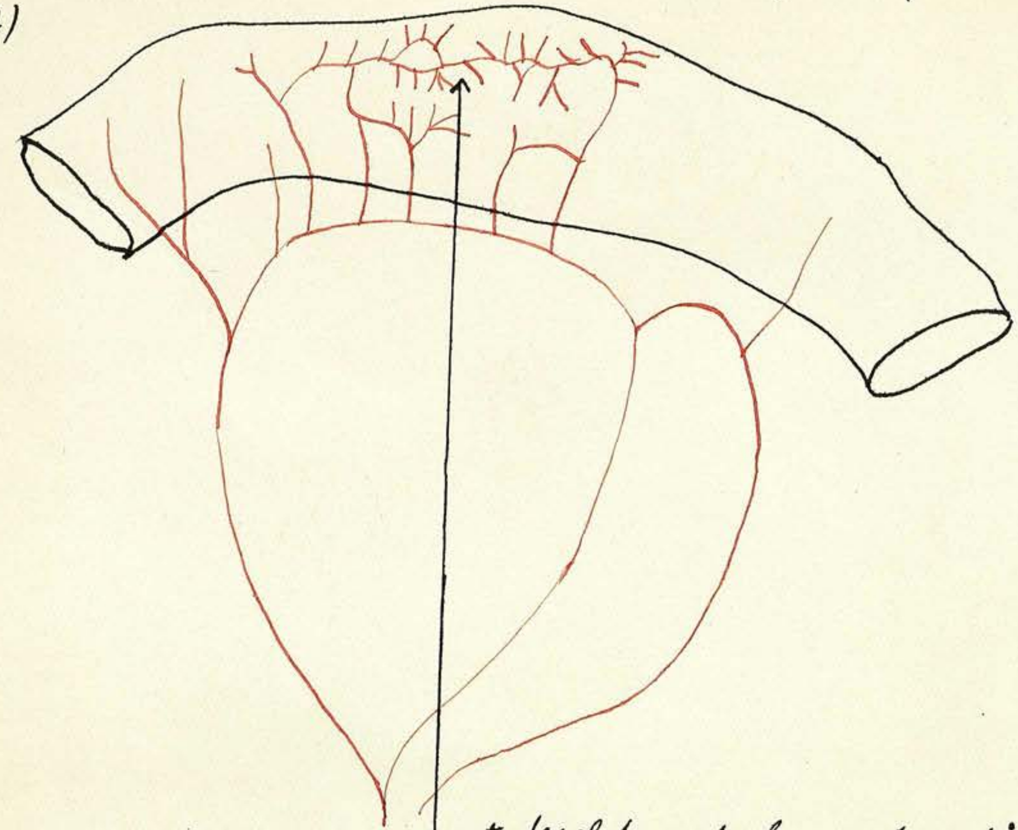
The cast was photographed. The main vessels are well seen; the injection material had not passed into the finer vessels.

Note the constant anastomosing channel across bifurcating vessels, producing the arcades in the mesentery.

No. 40 (Drawing)/

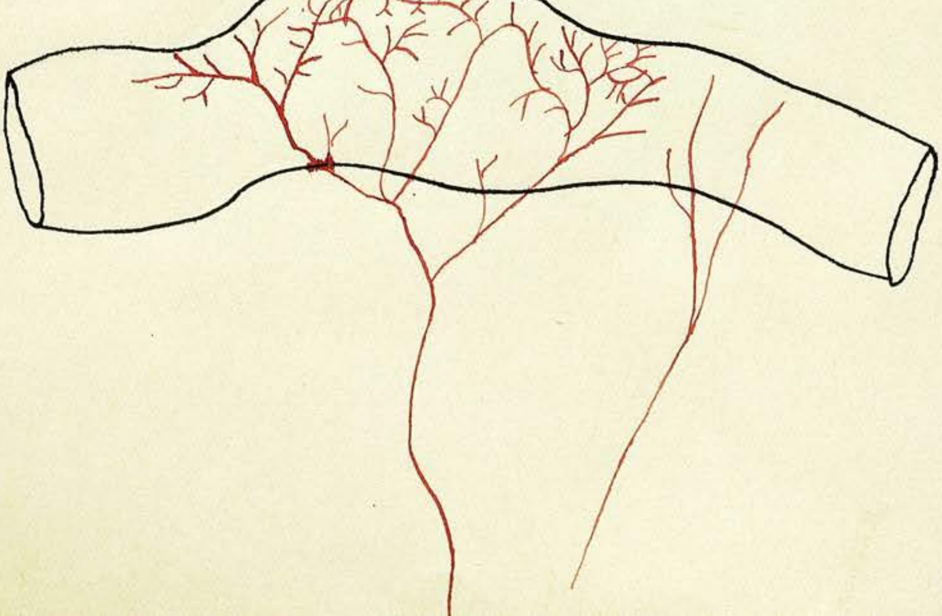
mid jejunum from an adult dog. 2 segments.

(A)



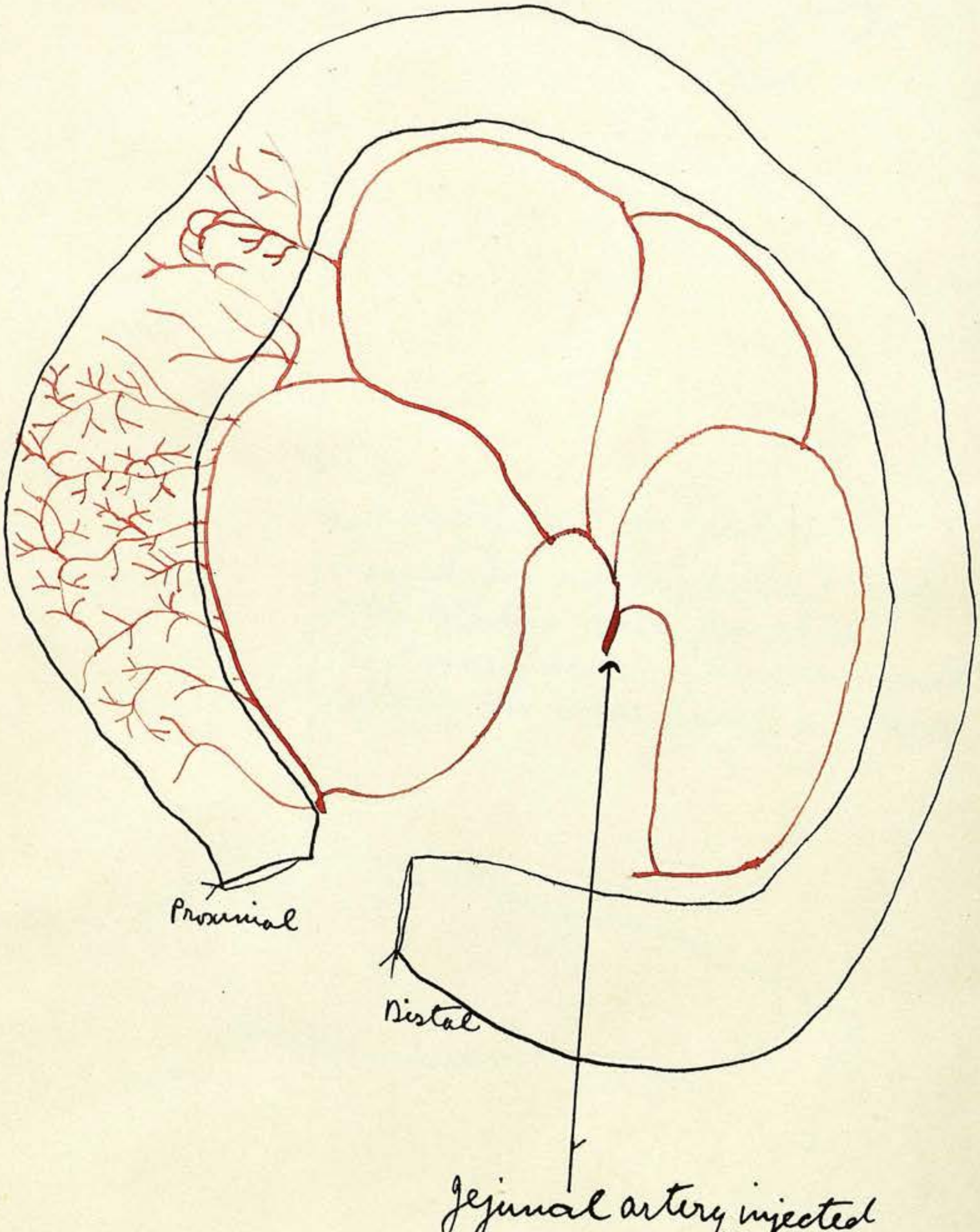
note development of anastomotic channels forming longitudinal vessels on the intestine wall from which short vessels arise. This arrangement replaces the vasa longa in places.

(B)



no 40

mid jejunum from an adult dog.



Nos. 40 and 41 (Drawings)

Specimen of mid jejunum from adult dog.

Specimen inflated with air and drawn.

Note the irregular size, direction and length of the vasa recta.

Note the great development of anastomotic channels on the gut wall forming longitudinal vessels from which short vessels arise. This arrangement replaces the vasa longa in considerable areas.

Conclusions/

CONCLUSIONS

As a result of these investigations certain observations can be made.

The arteries of the intestines follow a definite pattern. In the jejunum the mesenteric vessels form several arcades. These increase in number and complexity distally as the intestine is followed down into the ileum. There is no sharp demarcation, but a portion of upper jejunum can be readily recognized from a portion of lower ileum. An explanation for this complex system of vessels has been given by Franklin (1938), who considers it is for the accommodation and storage of blood - a blood depot system. The arrangement of arcades, with anastomotic channels across the bifurcations must also be related to the movements of the intestines. The pattern of arcades is an adaption to secure the passage of blood to the vessels nearer the intestine without interruption during peristaltic waves.

This system of arcades is comparable with the anastomotic channel between the two umbilical arteries in the placenta as has already been/

been described (Section I). Further evidence for this explanation is seen in the large intestine. In the more fixed parts of the large intestine, e.g. the descending colon, there is a main longitudinal trunk fed by branches of the mid and left colic arteries, the marginal artery of Hamilton Drummond. When a more mobile portion of the colon is reached, arcade development occurs again. This arrangement, noted also by Stewart and Rankin was seen repeatedly in the present series of specimens, the arcade formation between the sigmoid arteries being present in the pelvic colon region, whereas in the descending and "iliac" regions a single marginal artery was found.

Coming off the main vessels, that is the arterial arcades of varying complexity in the small intestine, the marginal artery in the ascending and descending colon and the arterial arcades of simpler formation in the transverse colon and pelvic colon, are the vasa recta. These vessels run to the intestinal wall, and proceed on it in line with the fibres of the circular muscle coat at right angles to the marginal artery in the colon; a secondary system of arcades is present to a variable extent between the roots of these vessels.

The/

The vasa recta are all of a similar size in the small intestine. In the colon they can be further classified as vasa longa (long), vasa brevia (short), and vasa intermedia (intermediate in size). The origin of these vessels has been noted in the different specimens examined:

(a) coming off singly to alternate sides of the colon

(b) coming off a common trunk of variable length, and branching to the same side of the intestine,

or

(c) to opposite sides.

In the jejunum, anastomoses between the roots of these vessels is infrequent, and the vessels run, without any union with their neighbours, direct to the gut wall. Here they give off a series of short vessels and branch repeatedly, forming free communications with their neighbours. Erroneous impressions can be formed about the vasa recta of the small intestine from observations of imperfectly injected specimens, and such statements made as "the vasa recta of the small intestine are end arteries" are quite inaccurate. In a well injected specimen (jejunum 1) the vasa recta form a perfect series of rings round the intestine, there being formed complete arterial circles round the gut by the junction of vessels from the opposite sides. This/

This arterial pattern is ideally adapted to preserve the free flow of arterial blood to the intestine in the presence of the contractions going on all the time in the muscle of the intestine wall and to expedite the venous return. There is very free anastomoses between the vasa recta on the wall of the small intestine, by fine vessels branching from them to meet their neighbours. (This anastomosis is so free that it is evident that even in extreme distension there will still be adequate blood supply to the small intestine).

The small intestine is therefore an extremely vascular structure.

In contrast to the small intestine, the large intestine is comparatively avascular. The origin of the vasa recta has been described above. There is not nearly as free communications between the vasa recta of the colon as on the small intestine. An imperfect tertiary arcade system is developed in places, on the colon wall, but cannot compare with the abundant connections of the vasa recta in the small intestine. There are anastomoses across the anti-mesocolic border between vasa longa of opposite sides, but these are infrequent, even in the specimen with the most penetrating injections. It is evident, therefore that the colon cannot withstand distension as well as/

as the small intestine; this comparatively poor blood supply explains the giving way of stercoral ulcers above an obstructing colonic carcinoma, and the tending of surgical entero-anastomoses of the colon to leak unless pressure on the suture line is relieved by a safety valve caecostomy or colostomy above.

The vasa recta run a straight or sinuous course in the gut wall, and begin to pierce the muscle coat in the colon just before reaching the taeniae coli.

No observation can be made in corrosion specimens regarding the point on the circumference where the vessels pierce the muscle wall; this has been investigated fully by Noer (1943) using liquid latex injections. The relation of the vasa recta to the appendices epiploicae was investigated, the base of several appendices epiploicae being ligated prior to injection. Though a slight bend in the vessel was present in some instances, there was no actual loop noted in any of the 26 colons examined, and in every instance the injected material flowed on distal to the ligature.

In some specimens the vasa longa

were/

were very tortuous, and an appearance very like a series of loops produced; but this was produced by the shrinking of the specimen on insertion into strong HCl, and could not be regarded as a true finding. Meillerè's work was not confirmed in this present investigation. A bend, or loop, on the course of a vessel of the intestine has the same principle as the tortuous vessels of the uterus - to allow for expansion of the organ they supply, and extension of themselves; it is therefore a formation which is consistent with the rules guiding the course of arteries, and should not be regarded as abnormal when encountered.

In the specimen of small intestine of a dog the most notable feature was the frequent presence of longitudinal anastomosis between vasa recta on the intestine wall, a feature which we have seen is by no means frequent in the human intestine, "tertiary arcades" between vasa longa only occurring three or four times in each specimen examined. Interpretations on the canine vascular pattern cannot therefore be applied to man.

The origins of the vasa longa from the marginal artery of the colon - some arising singly, some arising as a pair or even three off a common stem - may at first sight seem haphazard, but these are really insignificant variations on one theme/

theme and are perfectly normal - any area of intestine wall receives the same blood supply as its neighbour, though the course of the vessels going to it may vary.

The alterations in the arterial pattern of different regions of the intestine - vascular rings in the jejunum, plexiform arcades in the ileal mesentery-marginal artery in the descending colon - anastomotic channels across bifurcations of arteries - arcade formation reappearing on the pelvic colon - occur in a perfectly orderly and regular fashion throughout.

Slight variations may occur, but real abnormalities must be rare, None were detected in this series. The vascular pattern of the intestine appears well suited to the functions of this organ, both as regards the work of digestion and the mechanical movements of the organ.

SECTION IV/

SECTION IV

Muscle Arteries

In the previous section we have seen that the intestinal arteries are arranged so as to protect against any interference with the circulation caused by contraction of the musculature of the intestine. The study of the arterial pattern in skeletal muscle naturally follows, to see how the course of arteries are adapted to organs whose primary function is contraction.

The vascularisation of muscles has recently been brought under review again, in relation to the etiology of gas gangrene in war wounds. Wood Power (1945) noted that when one artery to a muscle was injured, then that part of the muscle supplied by the injured vessel became ischaemic when this occurred, and the re-establishment of the collateral circulation within the muscle appeared to be extremely slow. He described a case of complete gangrene of the long head of biceps following the severing by a shell fragment of a branch of the brachial artery which having passed through the short head of the biceps entered the long head.

Campbell and Pennefather (1919) studying the blood supply of muscles with special reference to war surgery, injected the main vessels of the region with a light bismuth salt and then radiographed the excised muscles. They divided muscles into three main classes:

(1) Those with a blood supply derived from many different sources and in which potential anastomosis between the different sources are quite numerous. The deltoid pectoralis major, pectoralis minor, the subscapular muscles, biceps, brachialis triceps, adductor magnus, gluteus medius, gluteus minimus, are given as examples of this class.

(2) Those with a blood supply derived from only two or three different sources, but in which the potential anastomosis between these sources are, relatively speaking, few in number. Examples:- gluteus maximus, rectus femoris, hamstring muscles, sartorius.

(3) Those with a blood supply derived, for all practical purposes, from only one source, and in which, granted that this main source of supply is cut off, almost the entire muscle becomes ischaemic and therefore liable to practically complete absence of potential collateral channels. Examples:- crureus, gracilis, gastrocnemius.

They stress the importance of conserving the blood supply to all muscles, as muscles rendered ischaemic by damaged blood supply are liable to gas gangrene, and noted that in the classical posterior route for excising the head of the femur the incision was planned to divide the gluteus maximus muscle approximately along the line of separation between the/

the portions supplied by the gluteal and sciatic arteries. Campbell and Pennefather wrote "we are justified in applying the term end-arteries to those inside muscle".

Le Gros Clark and Blomfield (1945) have studied the efficiency of intra muscular anastomoses, and observed the regeneration of devascularised muscle in rabbits using intravitam stain. They note that when all the main vessels of supply to a muscle are interrupted, part of the muscle can still be to some degree vascularised through small vessels which enter it at the site of its attachment. The extent to which this can occur naturally varies with the type of attachment. In broad fleshy attachments an effective blood supply can be obtained from this source. In thin membranous attachments vascularisation only extends into the muscle tissue immediately adjacent to the tendon. They note that "the ease with which it is possible experimentally to effect a functional devascularisation of part of a muscle by occlusion of one of its vessels of supply is of practical significance, for it is clear that injury to a limb may in the same way cause devascularisation lasting for some days. If intramuscular anastomoses in human/

human muscles are no more adequate than in the rabbit, the same applies to man, and muscle thus affected is peculiarly susceptible to anaerobic infection. Thus in the prophylactic surgical treatment of wounds involving muscle, it is of importance that muscular tissue whose blood supply has been seriously affected should be removed. In order to do this effectively, some knowledge of the vascular patterns within the muscles of the human body is requisite so that the extent of the devascularised tissue can be approximately estimated. Unfortunately, little precise information is yet available on this subject".

Anrep in a series of papers (Anrep, Blalock and Samaan, 1934; Anrep, Cerqua and Samaan, 1934; Anrep and Saalfield, 1935) studied the effect of muscle contraction upon the blood flow in skeletal muscle. He found that contracted muscle affords a greater resistence to the arterial inflow than relaxed muscle. The effect of contraction of the skeletal muscle and of the intestine upon their circulation is due to a mechanical compression of the blood vessels by the contracting muscle fibres. A comparison of the venous outflow and of the arterial inflow of blood into a contracting muscle shows that muscular contraction is accompanied by a/

a compression of the intramuscular blood vessels. "The suggestion made by Rein that the blood vessels of a muscle present during contraction a diminished resistance to the blood flow cannot be confirmed".

What is the arrangement of the vessels then in this contractile organ? Krogh (1929) refers to the work of Spalteholz (1888) in this field. "The arteries supplying a muscle branch freely, and between the branches there are very numerous anastomoses forming a primary network. Into the meshes of this net small arteries are given off at regular intervals, and these again anastomose freely forming a secondary cubical net of great regularity. From the threads of this network the arterioles branch off, generally at right angles to the muscle fibres, and at very regular intervals (of about 1 mm in the warm blooded animal) and these arterioles finally split up into a large number of capillaries running along the muscle fibres and in the main parallel with them, but with numerous anastomoses, forming long narrow meshes about the fibres. The capillaries unite into venules intercalated between the arterioles, and the whole system of veins reproduces and follows almost exactly that of the arteries.

The/

The whole of the vascular system is beautifully adapted to the changes of muscle contraction; the arteries and venous networks ensure supply and drainage of almost every point, even if a number of anastomoses are temporarily blocked".

Blomfield (1945) has recently studied the intravascular patterns in man. He injected fluid barium into the finer vessels and then filled the larger arteries with a mixture of barium sulphate and celloidin. The main limb artery with the muscles supplied by it were then removed together in a single group and the whole preparation X-rayed. He describes five main types of intramuscular vascular patterns. These are:-

(1) A longitudinal anastomotic channel formed by a succession of separate nutrient vessels entering the muscle throughout most of its length; e.g. soleus and peroneus longus.

(2) A longitudinal pattern of vessels derived from a single group of arteries which rise from a common stem and enter one end of the muscle. e.g. gastrocnemius.

(3) Radiating pattern of collaterals which rise from a single vessel entering the middle part of the muscle; e.g. biceps brachii.

(4) A pattern formed by a series of anastomotic loops ranging throughout the length of the muscle and derived from a succession of entering vessels; e.g. tibialis anterior, extensor longus hallucis.

(5) An open quadrilateral pattern with sparse anastomotic connections.

(1) and (4) subdivisions appear to be practically the same; this will be discussed later.

A scrutiny of these different patterns leads to an enquiry why do these differences exist? A study of the morphology is not enough - some explanation must be sought. Do muscles, not necessarily developmentally the same, but with comparable function have a comparable blood supply? Do muscles whose fasciculi run in a similar way, have the same pattern of vessels? Are the blood vessels arranged with the same care as in the placenta and intestine with regard to the movement of the organ they supply?

Before starting an investigation into the blood supply of muscles, therefore, a knowledge of their morphology is required. Muscles have been classified with advantage for the determination of their mode of action, according to the arrangement of their component fibres (Quain 1923).

1. Muscle fibres forming plane surfaces

   A. The muscle fibres are parallel and rectilinear.

   (i)/

(i) Quadrilateral muscles, whose cross section remains constant over the whole length of the muscles. Thyrohyoid, Sartorius, for example.

(ii) Fusiform muscles, commoner than (1), taper at both ends. Fibres parallel, The area of transverse section remains almost uniform throughout the fleshy part of the muscle. Biceps brachii for example.

(iii) Rhomboidal muscles. These muscles consist of fasciculi which are parallel to one another, but pass with a greater or less degree of obliquity from origin to insertion. Semimembranosus for example.

Rhomboidal muscles are further subdivided into unipenniform, bipenniform, and multipenniform types. The bipenniform consist of two series of rhomboidal muscles attached, usually with equal angles of inclination to the opposite sides of a central tendon (Rectus femoris); multipenniform muscles are made up of several penniform muscles; the deltoid,for example, which is made up of seven distinct penniform muscles.

B. The muscle fibres are parallel and curvilinear.

Sphincter muscles. In sphincter muscle each fibre forms a curve, and the collection of such fibres parallel and in the same plane compose the entire muscle.

C. The muscle fibres are rectilinear but not parallel.

Triangular muscles. These have a wide origin, and converge to a narrow insertion, i.e. adductor pollicis or trapezius, or a pointed origin, and wide insertion, i.e. adductor longus.

2. The muscle fibres form curved surfaces.

A. The muscle fibres are curvilinear.

(i) Spheroidal muscles.
i.e. the muscles of the abdominal wall, and the diaphragm.

(ii) Ellipsoid muscles.

B. The muscle fibres are rectilinear

(i) "Skew" muscles, i.e. adductor magnus.

It was decided to select examples of certain of these groups, investigate them and see if the vascular patterns were similar in different numbers of the same group.

The muscles selected were:-

(1) The triceps surae muscle, selected in view of the different origins of its component parts.

(2) The sartorius and gracilis, examples of "quadrilateral" muscles with parallel rectilinear fibres.

(3) The biceps brachii, biceps femoris, and semitendinosus, examples of fusiform muscles.

(4) The deltoid, an example of a multipenniform muscle, and a similar muscle of the lower extremity, gluteus maximus.

(5) The sphincter ani, an example of a muscle with parallel, curvilinear fibres.

MUSCLE VESSELS

Triceps Surae

Experiments (1) and (2)

Specimen - Lower half of trunk of full term still-born male foetus, Foetus A.

Left common iliac artery injected with celloidin and acetone.

The cast was friable and broke up. It showed

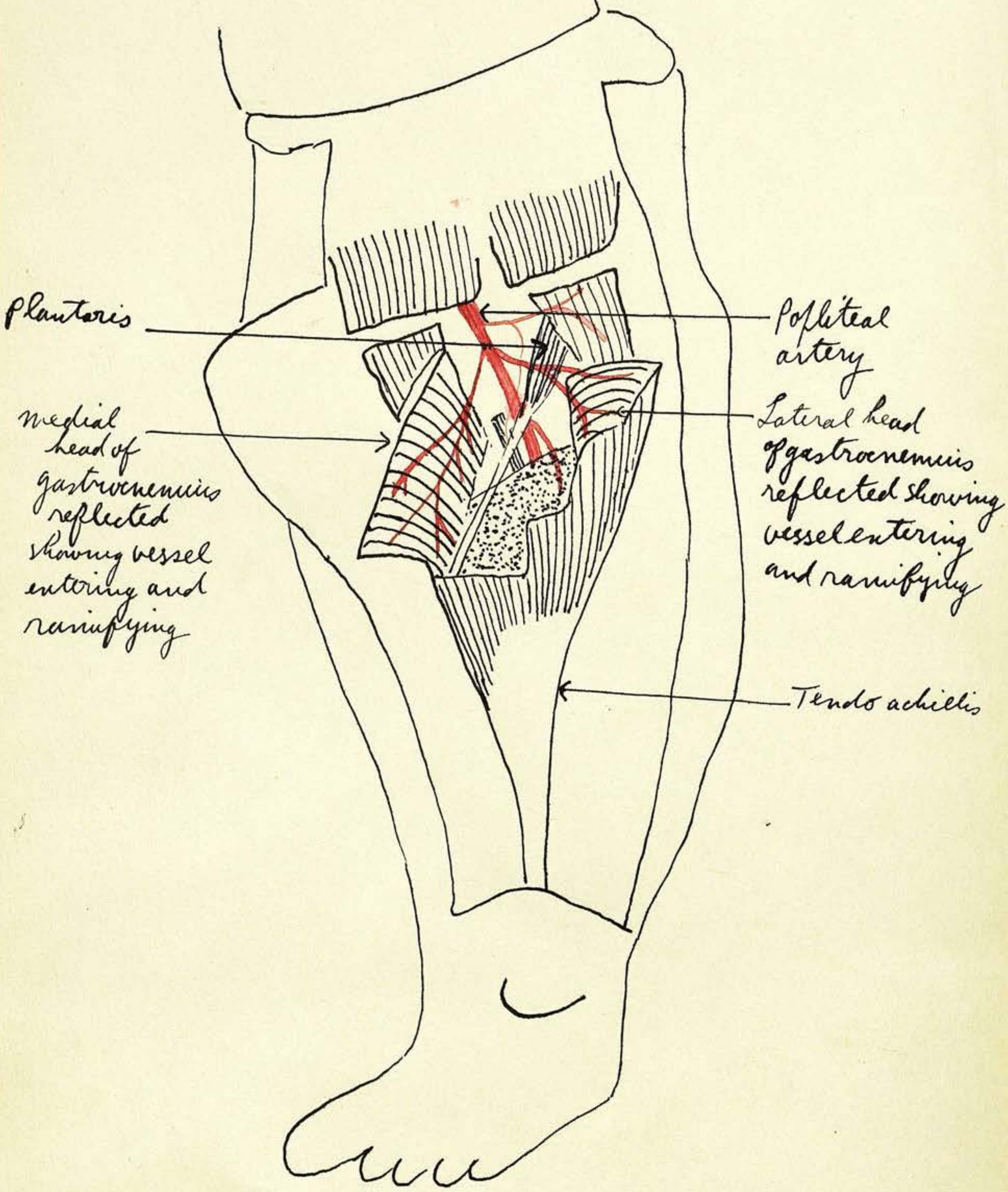
(1) vessels entering tendo achillis from post. tibial artery

(2) numerous vessels entering soleus from peroneal artery

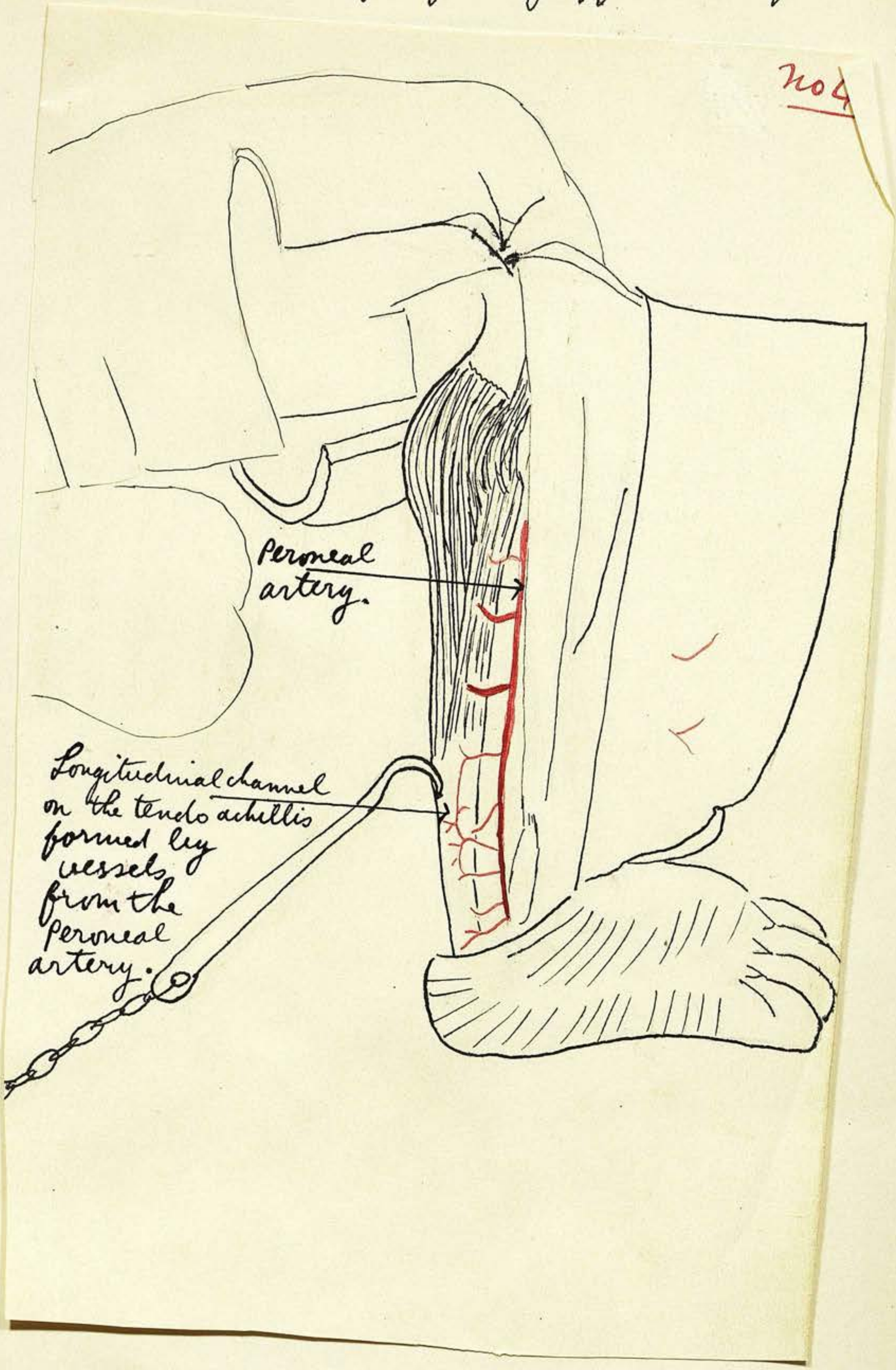
(3) vessels going to gastrocnemius, not anastomosing with (1) or (2).

Experiment (2)/

Rt. leg of full-term foetus



Lateral view of right leg of full term foetus



Experiment (2).

Nos. 41A and 42 (Drawings)

Specimen - Right common iliac artery of Foetus A.

Injected with a mixture of starch and red lead by hand syringe and then dissected and drawn.

This shows the arteries to gastrocnemius arising from the popliteal artery, and passing into the muscle from above, ramifying as longitudinal channels within the heads of the muscle.

No junctions were noted with the vessels of soleus which arose from the post.tibial and peroneal arteries and passed in as many single trunks into the fleshy origins of the muscle from the tibia and fibula.

The blood supply of the tendo achillis was well seen in this specimen, coming off as a series of vessels from the peroneal and the post.tibial arteries in sequence with the arteries supplying soleus, and being connected with each other on the tendon inside the tendon sheath by a series of anastomotic loops forming a longitudinal channel.

Experiment (3).

Specimen - Gastrocnemius soleus and plantaris with portion of popliteal and post.tibial arteries removed at Post Mortem from a female schizophrenic by Dr. W. Blackwood.

The cannula had been mistakenly inserted into the post.tibial artery, and consequently the arteries of gastrocnemius were not injected.

The vessels of soleus formed an intact cast, consisting of the posterior tibial artery, the peroneal artery, and vessels coming off them, ending without any connections at the margins of the muscle, or any vessels passing into the substance of gastrocnemius.

131

no 42A

Cast of Popliteal artery and
vessels of gastrocnemius and soleus

Popliteal artery

Vessels going to the 2 heads of gastrocnemius

Vessels going to soleus.

Posterior tibial artery

Peroneal artery

Experiment (4).

No. 42 A (Photograph)

Specimen - Triceps surae and accompanying vessels with portions of lower end femur and upper ends of tibia and fibula from which the muscle arose, from an adult patient who had died following operation for a cerebral tumour (Post Mortem by Dr. Blackwood).

Popliteal artery injected.

Photographed.

This specimen shows a part of the popliteal artery, continuing as the posterior tibial, with the peroneal artery.

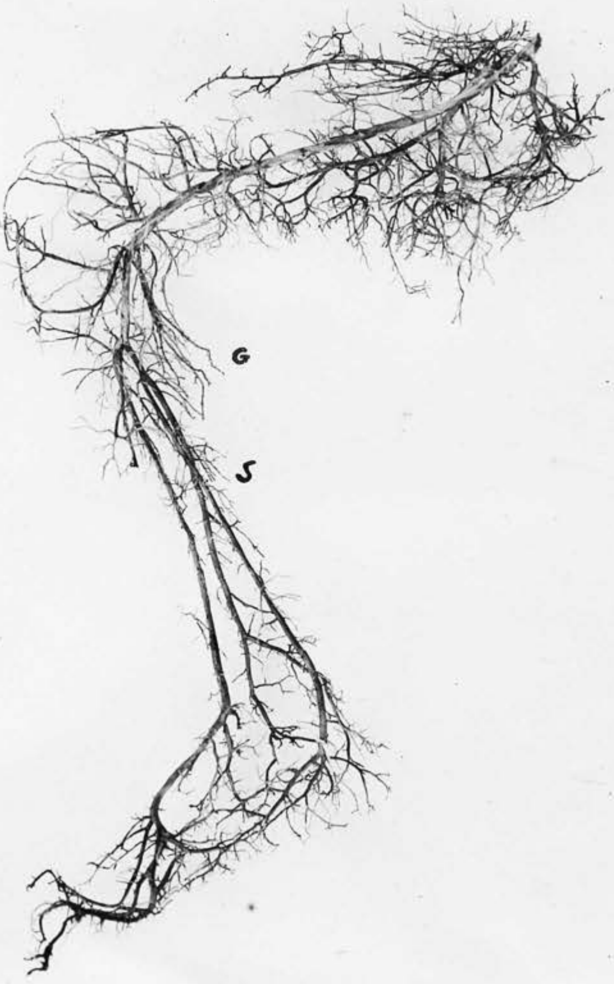
Two main arteries came off for each head of gastrocnemius, and two sets of vessels came off lower down from the posterior tibial and the peroneal, to the soleus.

No communication is present between these vessels, which appear to be end-arteries.

Experiment (5)/

no 43.

cast of vessels of right lower limb
of 5 day old infant.



G = vessels
of gastrocnemius
ending freely.

S = vessels of
soleus from
Posterior tibial and
Peroneal arteries

Experiment (5).

No. 43 (Photograph)

5 day old female infant, died of cerebral haemorrhage. Foetus B.

Specimen - Lower half of trunk.

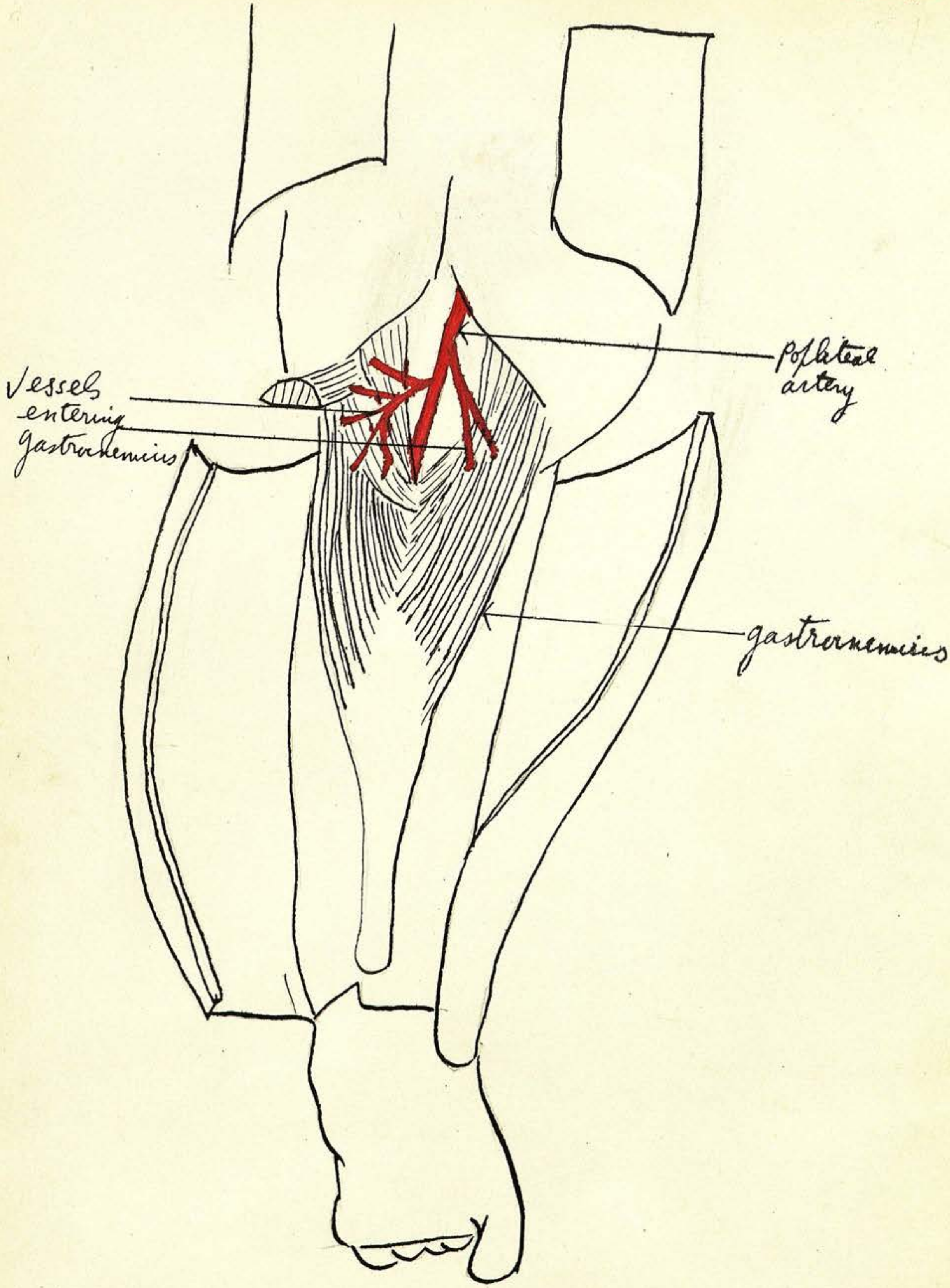
Satisfactory celloidin cast of vessels of right lower limb obtained.

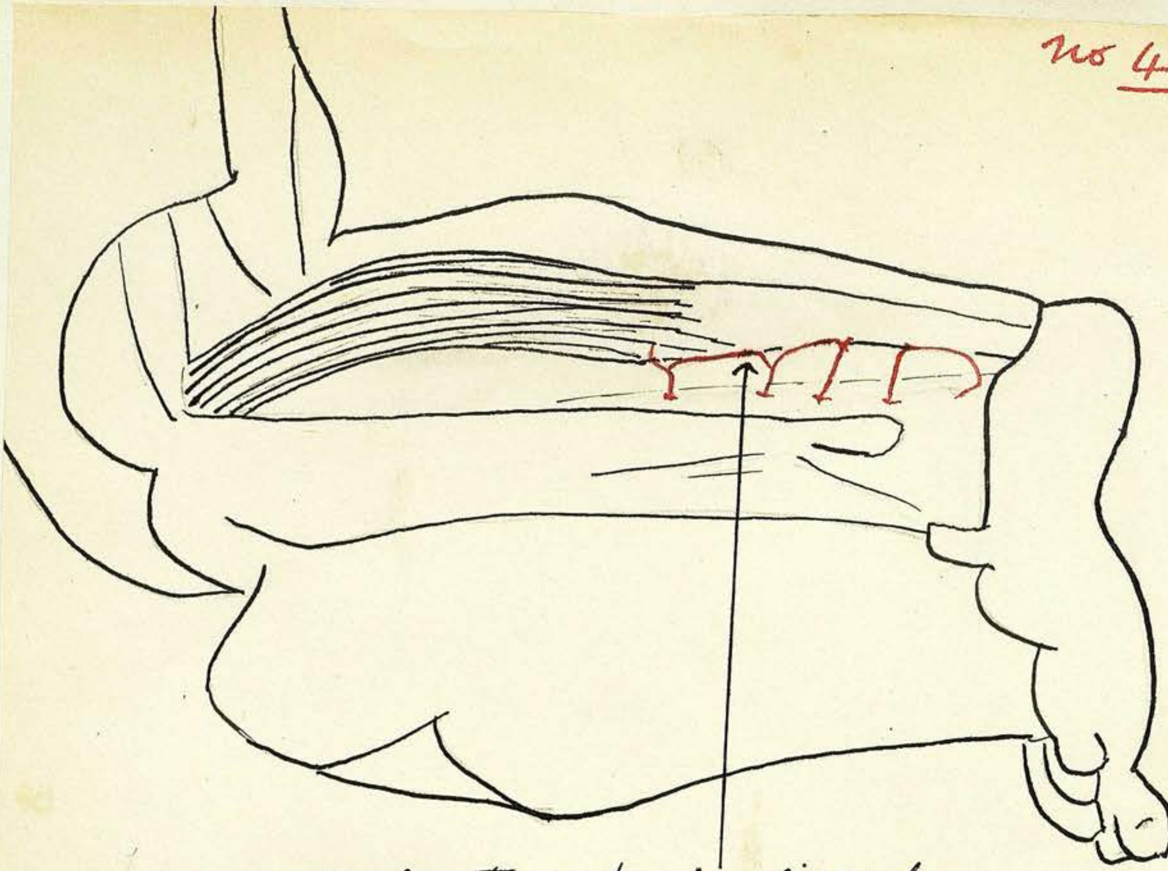
Photographed.

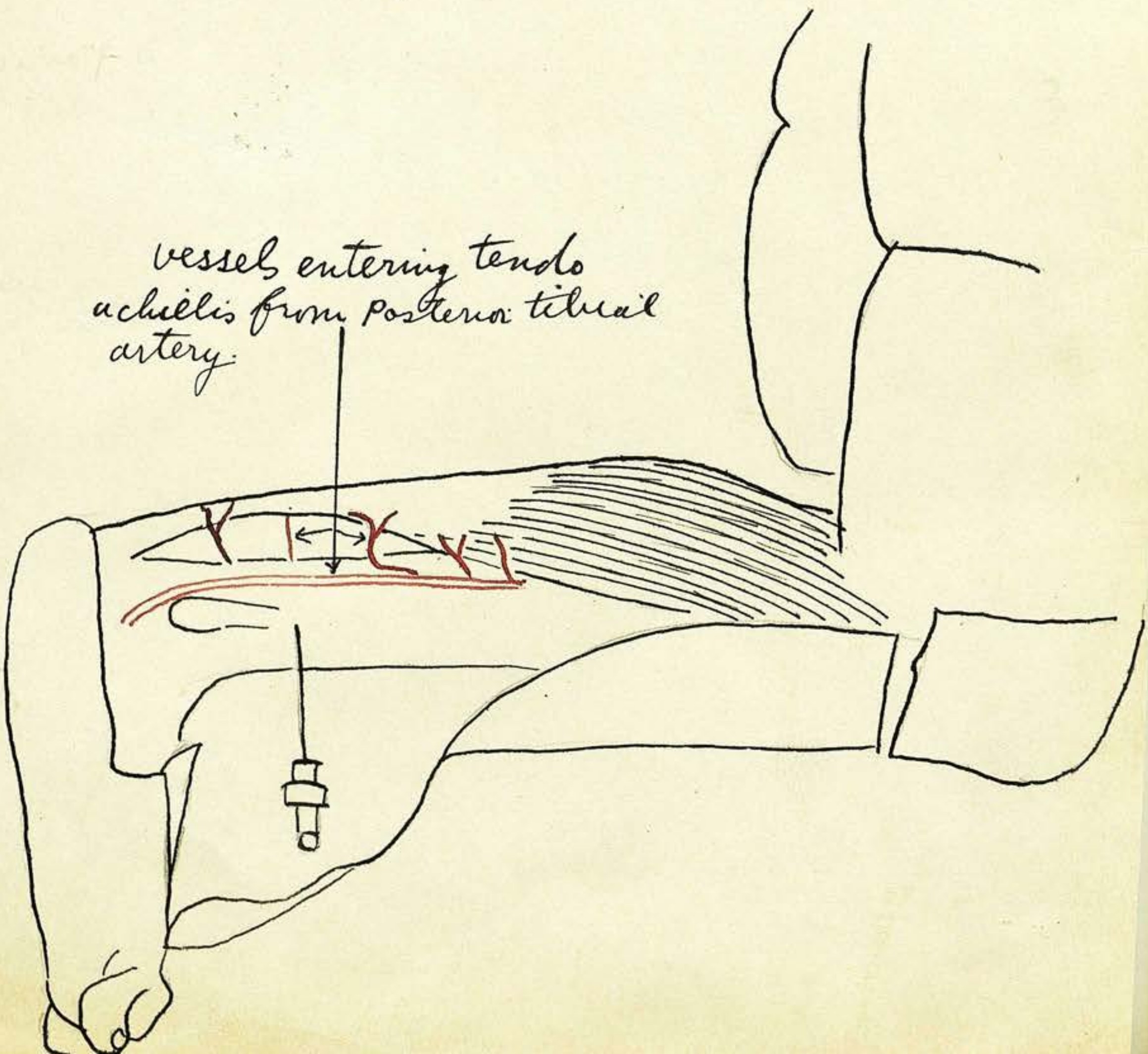
This cast shows the vessels of gastrocnemius arising from the popliteal, ending freely without any connection with the vessels of soleus, which came off in series from above downwards off the posterior tibial and peroneal arteries.

The soleal vessels are joined to each other at several places by slender longitudinal anastomosing vessels (not shown on photograph).

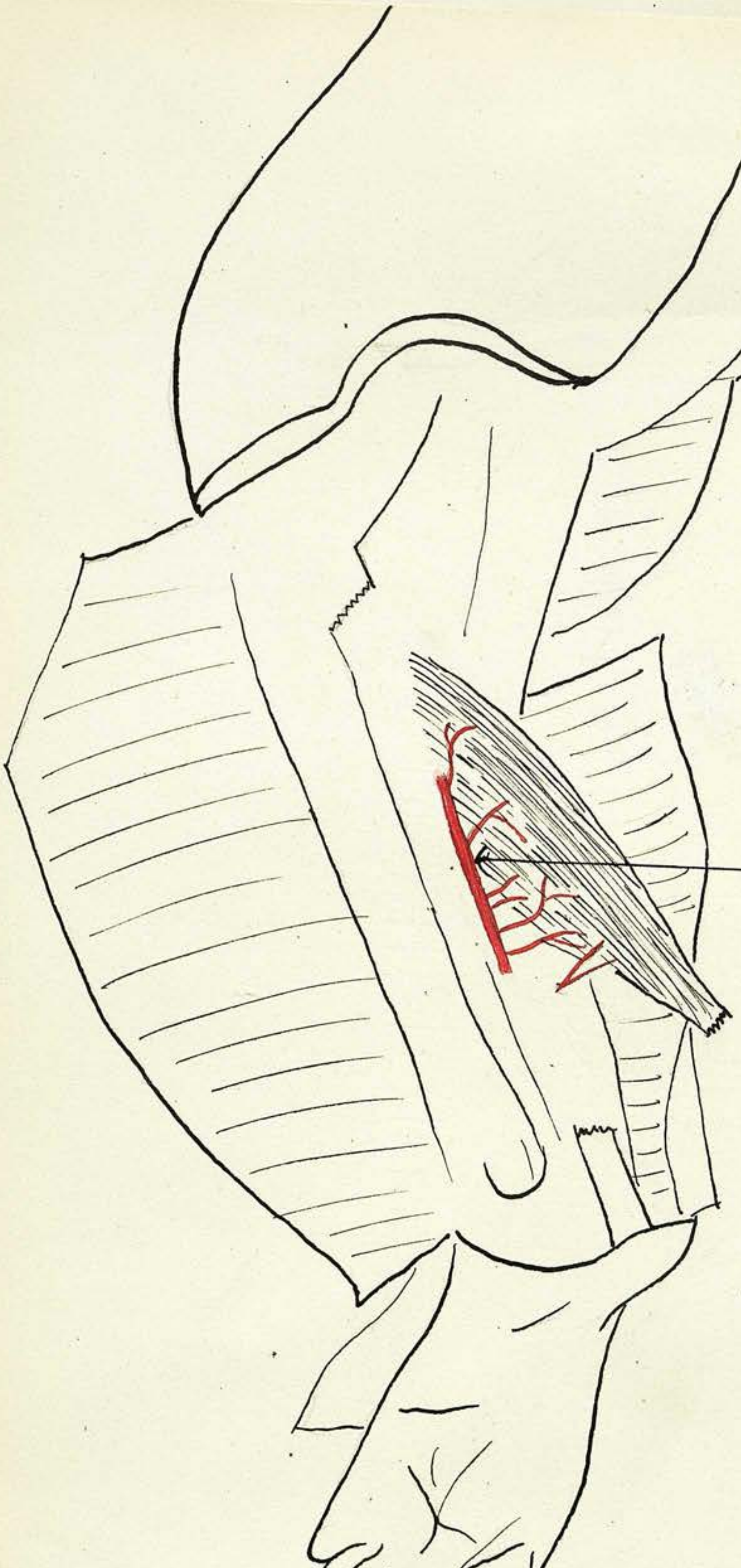
Experiment (6)/


Popliteal artery
Vessels entering gastrocnemius
gastrocnemius


vessels entering tendo achillis from
lateral aspect.

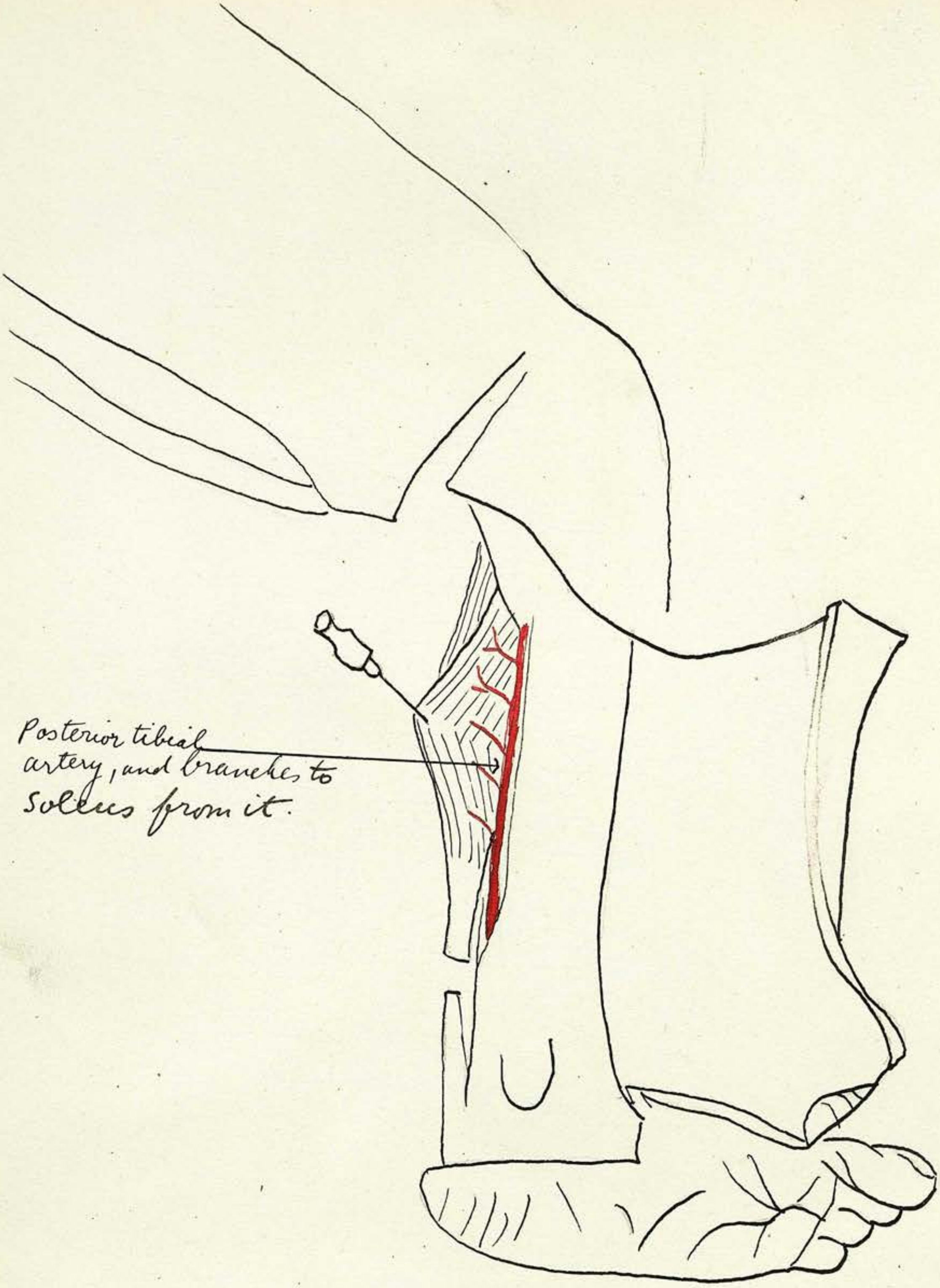

vessels entering tendo
achillis from posterior tibial
artery.

no 4



Peroneal artery
and vessels going from it
into soleus, the deep
aspect of which is
shown.

no 4



Experiment (6).

Nos. 44, 45, 46, 47 (Drawings).

Specimen - Left external iliac artery of Foetus (B), injected with starch-red lead solution, by hand pressure syringe, and then dissected.

Drawings:

44 shows vessels entering gastrocnemius from above.

45 the vessels entering the tendo achillis from the lateral side, peroneal artery,

and

entering the tendo achillis from the medial side, posterior tibial artery.

46 shows the vessels supplying soleus from the peroneal artery.

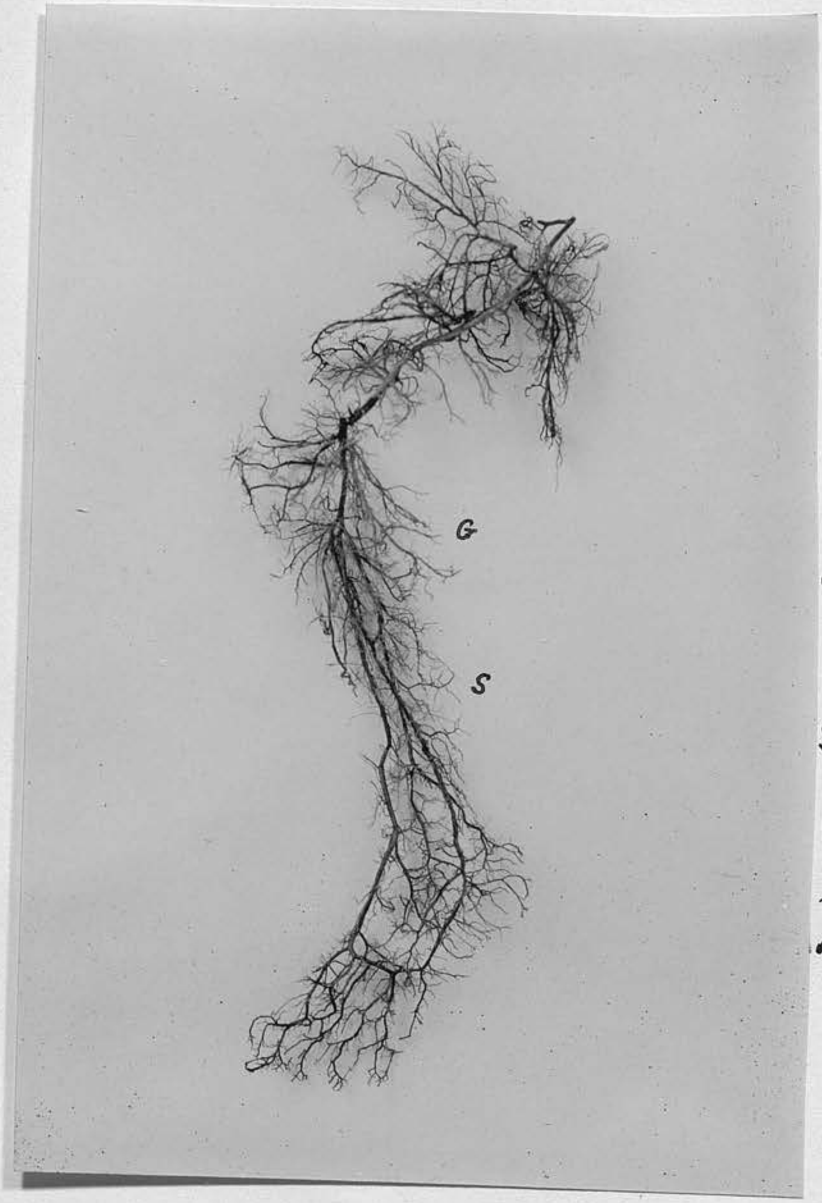
47 the soleal vessels from the posterior tibial.

No connection between the gastrocnemius vessels and those of soleus were demonstrated. The vessels entered soleus in its thick fleshy origin, ramifying parallel with the muscle fibres, and being connected by slender longitudinal channels in places - this being seen best on the tendo achillis in 45.

Experiment (7)/

no 48

Cast of vessels of right lower limb
of full term foetus



G = vessels for
gastrocnemius
arising from
popliteal, and
ending freely.

S = vessels to soleus
from post. tibial
and peroneal arteries,
joined to each other
by longitudinal
anastomosing
vessels.

Experiment (7)/

No. 48 (Photograph)

Still-born female infant, died of cerebral haemorrhage. Foetus (C).

Specimen - Lower half of trunk.

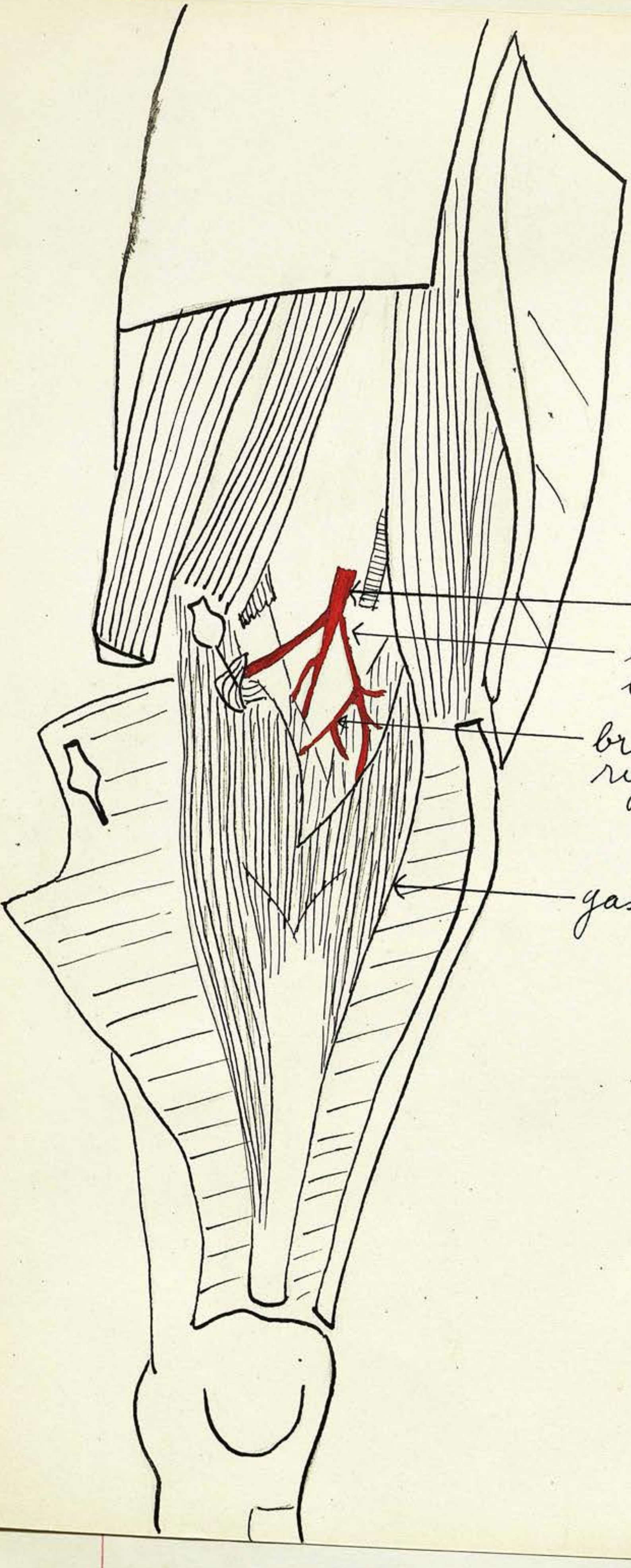
Satisfactory celloidin cast of right lower limb obtained.

Photographed.

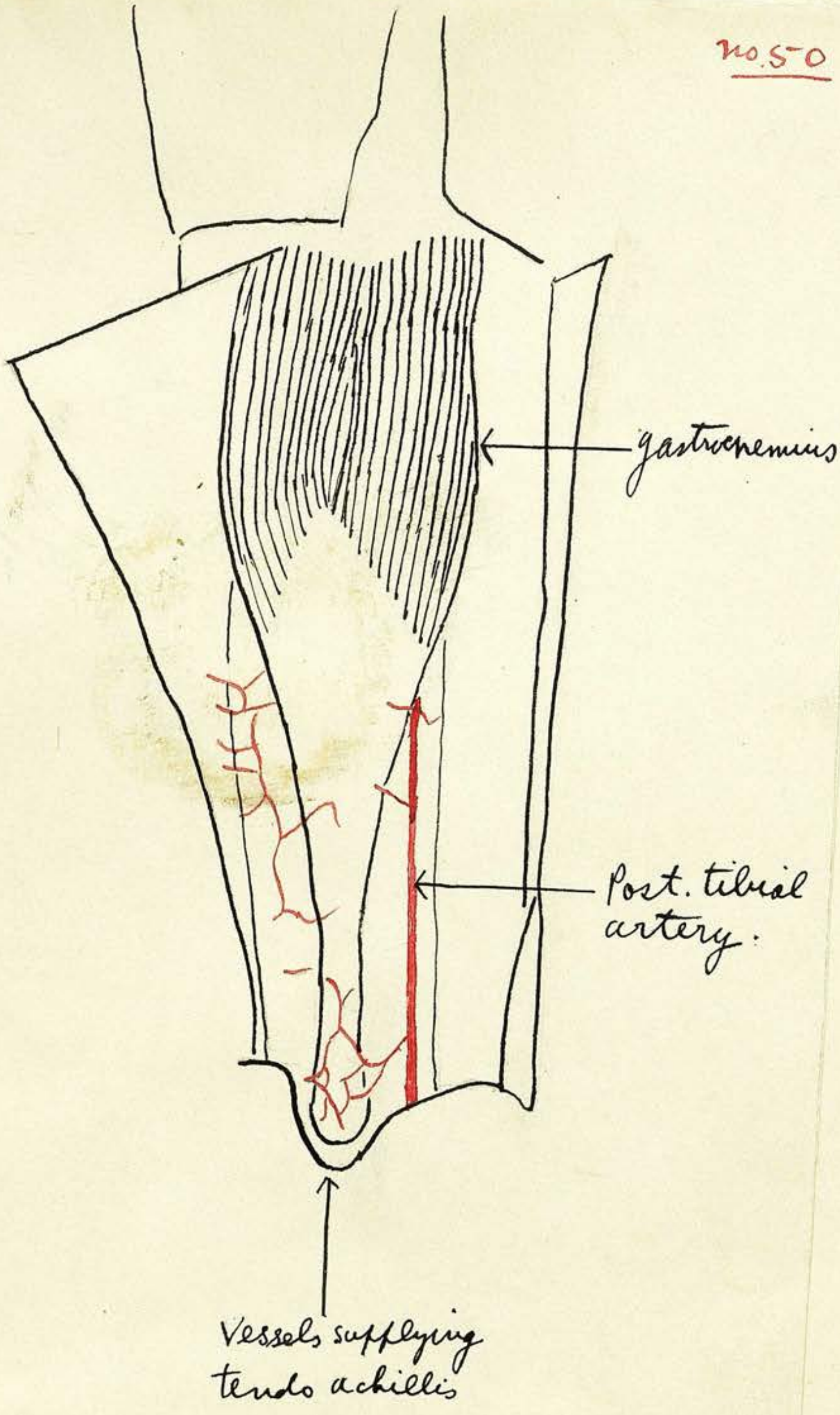
The specimen showed the vessels of gastrocnemius arising from the popliteal artery, ending freely without any connection with the vessels of soleus.

The vessels of soleus came off in series from above downwards, off the posterior tibial and peroneal arteries, and are joined to each other at several places by slender longitudinal anastomosing vessels.

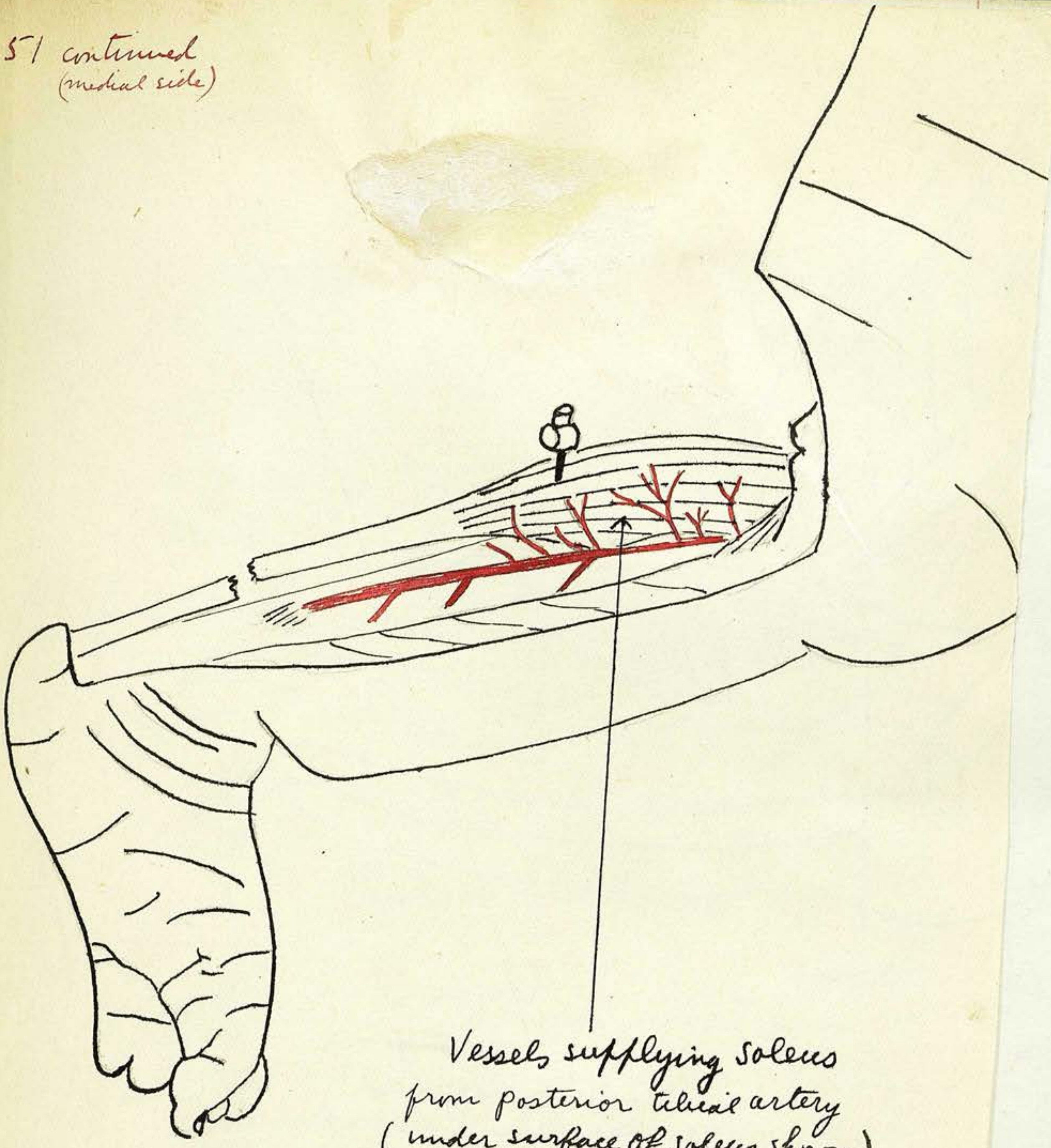
Experiment (8)/


Popliteal artery
branches to medial and
lateral heads of gastrocnemius
branch to left head from
right artery
gastrocnemius.

no. 50

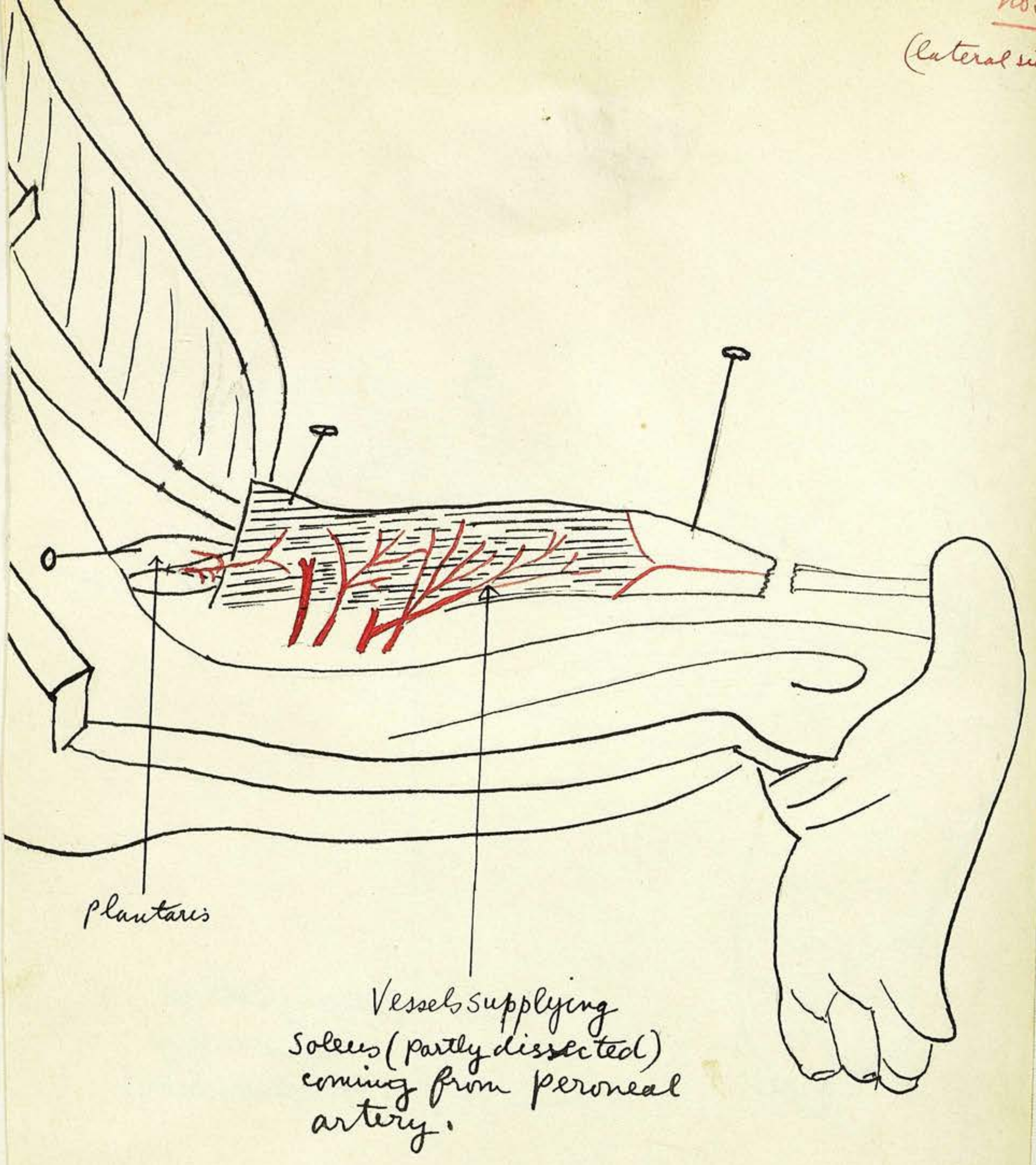

gastrocnemius
Post. tibial artery.
Vessels supplying tendo achillis

51 continued
(medial side)



Vessels supplying soleus
from posterior tibial artery
(under surface of soleus shown)

(lateral side)



Experiment (8).

Nos. 49, 50 and 51 (Drawings)

7.1.46. Left external iliac artery of Foetus (C) injected with starch red lead solution by hand pressure syringe and then dissected.

Drawings:

49 shows vessels entering gastrocnemius from above, a branch from the right artery going to the left head.

50 vessels supplying the tendo achillis.

51 the arteries supplying the muscular part of soleus.

Note the arrangement - a series of vessels coming in and being joined in places by longitudinal anastomotic channels, which join across the mid line on the tendo achillis.

No connection demonstrated between vessels of gastrocnemius and soleus.

Experiment (9)/

nos 5-2,

Photograph of xray, lateral view, of adult right leg (amputation specimen) injected with red lead-barium sulphate mixture.

Popliteal artery

vessels to gastrocnemius

vessels to soleus.

no 53

Photograph of Xray, A.P. view, of adult right leg (amputation specimen) injected with red lead - barium sulphate mixture (Same specimen as no 52)

Popliteal artery

Experiment (9).

Nos. 52 and 53 (Photographs)

Specimen - Right leg of an adult female, amputated through the lower third of the thigh for old poliomyelitis by Mr. Rutter on 4.1.46.

Popliteal artery perfused with water. Then vessels perfused with water again and injected with a suspension of starch red lead and barium sulphate, in the proportion of starch and red lead : barium sulphate, 4 : 1. Leg then placed in preservative fluid consisting of glycerine, formalin, carbolic and methylated spirit.

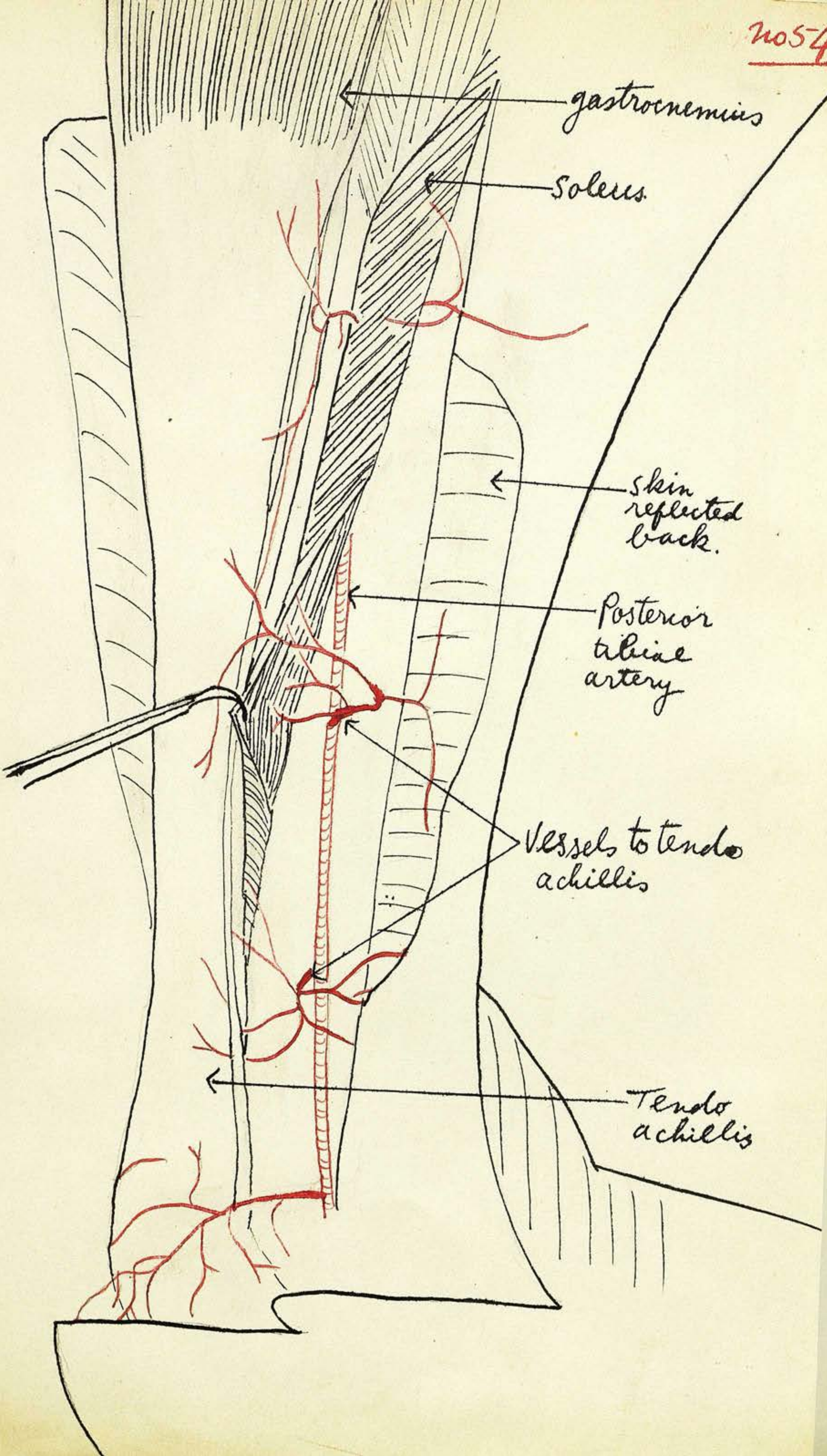
X-rayed. Films photographed. Antero-posterior and lateral views.

The photographs are reproduced from the X-ray negatives, reduced on to a half plate and contact prints taken. The photographs show part of femur, tibia, fibula, popliteal artery, and arteries of leg. The vessels of the gastrocnemius are seen arising from the popliteal, with slender vessels passing down near the surface. Numerous vessels are seen arising from peroneal and posterior tibial arteries, passing into soleus and running down longitudinally in the line of the muscle fibres.

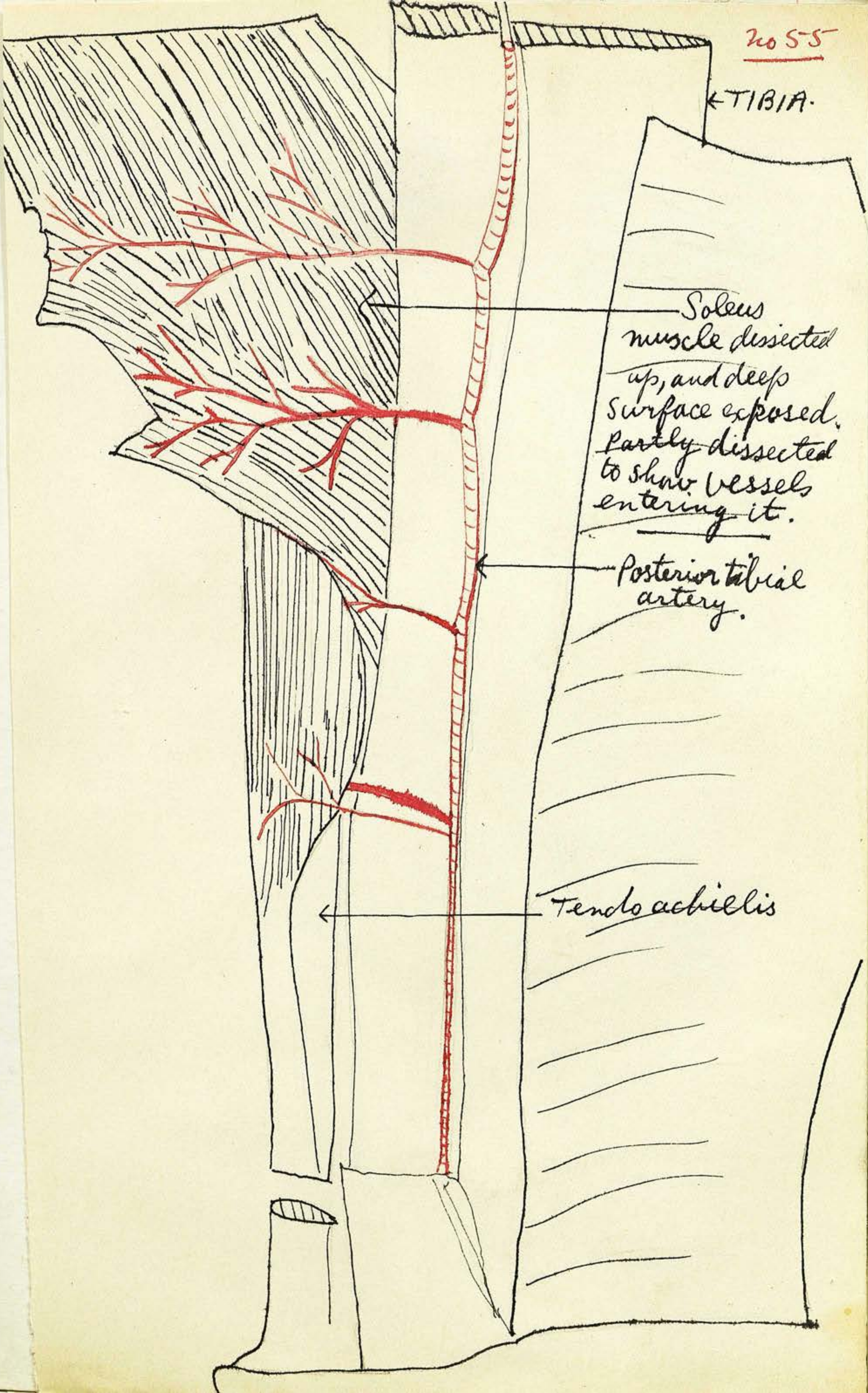
When viewed stereoscopically, these vessels are seen as if clasping the soleus and tendo achillis in a series of vascular rings, anastomosing in places with the slender vessels descending longitudinally from the gastrosnemius' arteries.

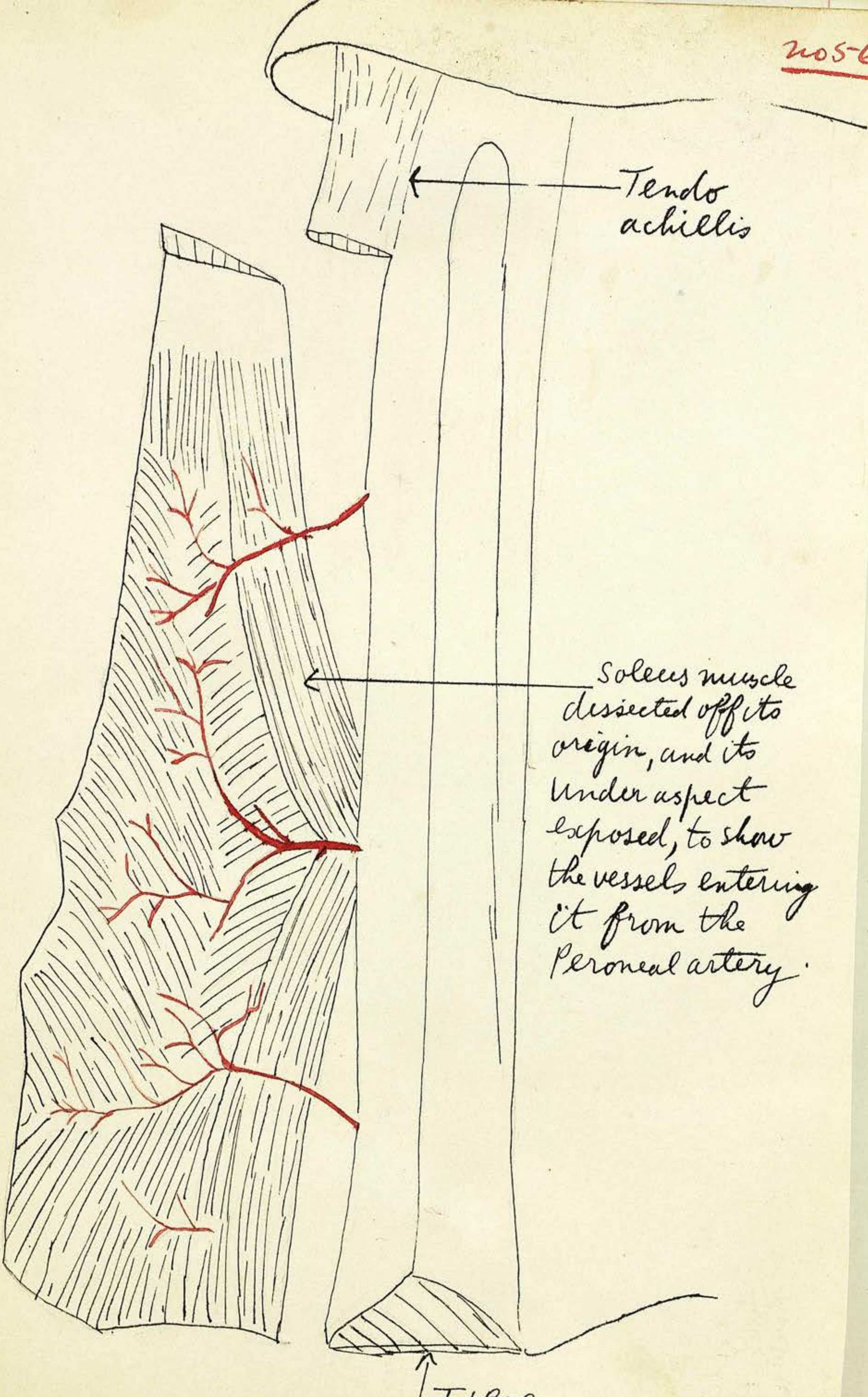
Incidentally, the anastomosis around the knee joint is well demonstrated.

no54


gastrocnemius
Soleus.
skin reflected back.
Posterior tibial artery
Vessels to tendo achillis
Tendo achillis

no 55


TIBIA.
Soleus muscle dissected up, and deep surface exposed. ~~Partly~~ dissected to show vessels entering it.
Posterior tibial artery.
Tendo achillis


Tendo achillis
Soleus muscle dissected off its origin, and its under aspect exposed, to show the vessels entering it from the Peroneal artery.
TIBIA.

Experiment (10)

Nos. 54, 55 and 56 (Drawings)

Specimen - Adult leg from junction of upper-mid third from an amputated limb, reason for amputation unknown.

Posterior tibial artery perfused with water then injected with vermilion compound (Ferguson) via hand pressure syringe.

X-rayed. Unsatisfactory X-ray result - vessels appeared fragmented.

Specimen dissected and drawn.

Drawings:-

54 shows the vessels going to the tendo achillis from the posterior tibial artery, forming longitudinal anastomotic channels. These vessels were not so large in proportion to the size of the tendon as in the foetal specimens, previously seen.

55 shows the vessels entering and ramifying in soleus from the posterior tibial,

and

56 from the peroneal artery.

The finer ramifications were in line with the muscle fibres.

Experiment (11)/

no 57

Cast of vessels of left lower limb of full term foetus, showing the arterial pattern.

F = femoral artery

S = longitudinal anastomosis between vessels of Soleus.

Experiment (11).

No. 57 (Photograph)

Full term still-born foetus died of cerebral haemorrhage. Foetus (D).

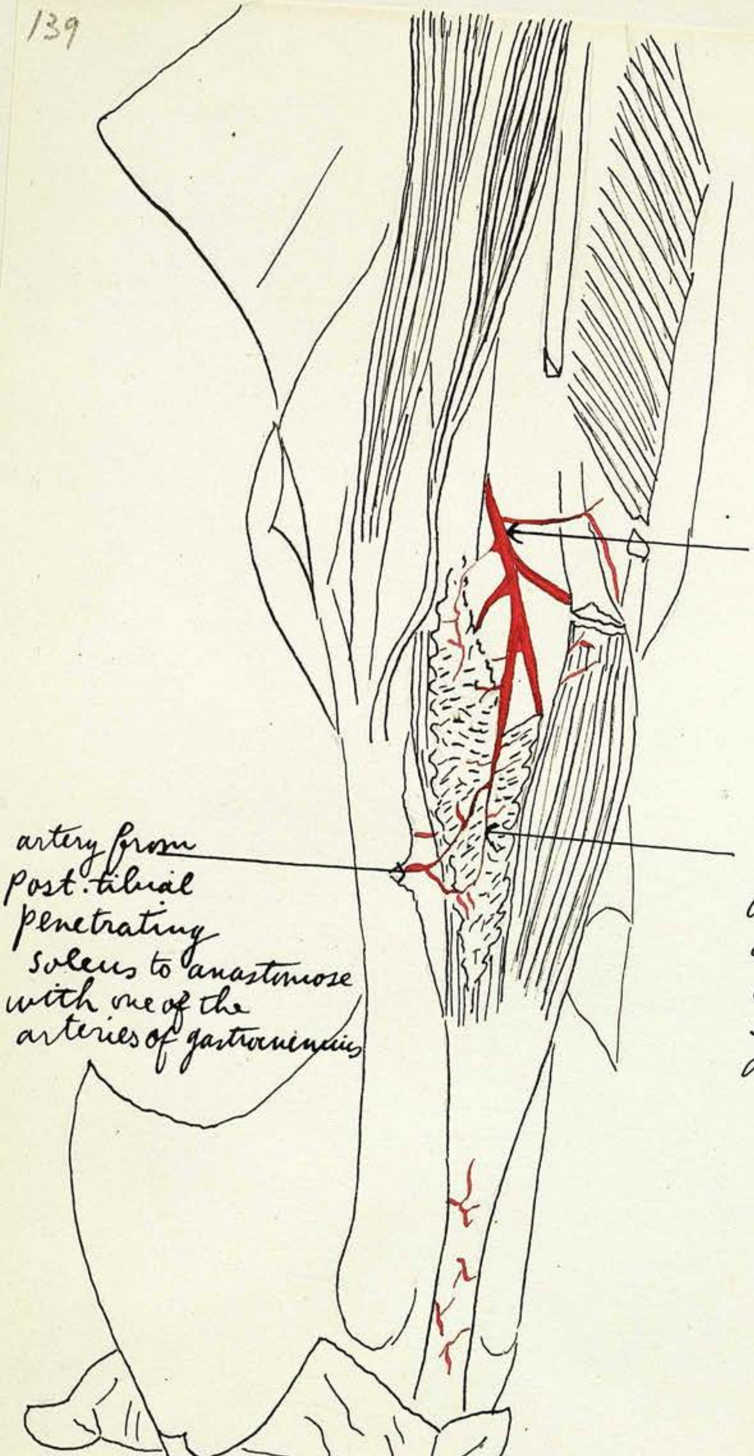
Satisfactory celloidin cast of arteries of left lower extremity.

Photographed.

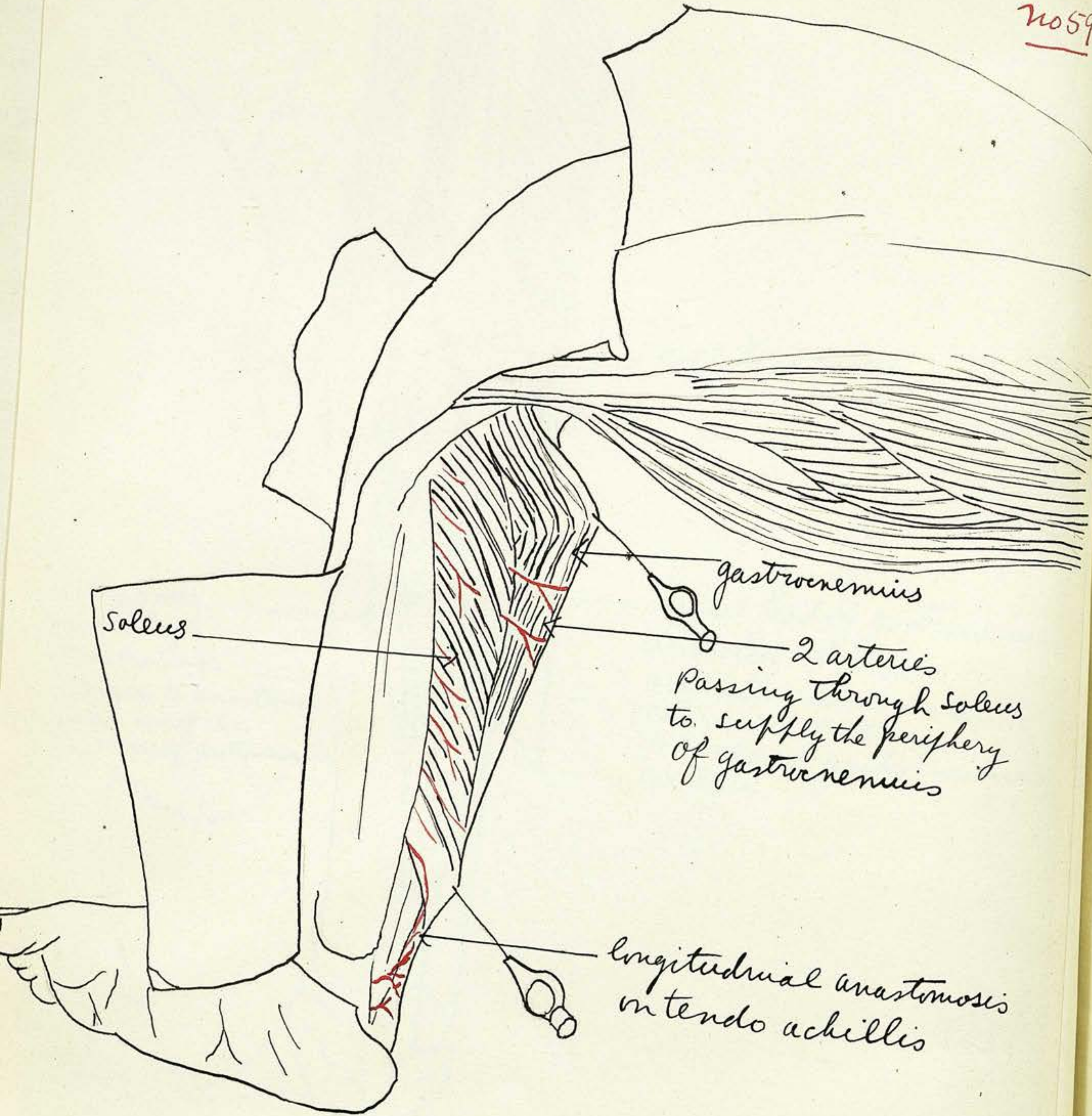
This cast shows the arteries of the lower extremity in fine detail. The arteries to gastrocnemius were seen entering from above, and one slender vessel connected the artery in the lateral head of gastrocnemius with a soleal artery from the porterior tibial. The longitudinal anastomoses between the soleal arteries was well developed, and can be seen in the photograph.

Experiment (12)/

no 58


Popliteal artery
artery from Post. tibial penetrating soleus to anastomose with one of the arteries of gastrocnemius
Medial head of gastrocnemius dissected to show arterial anastomotic channels between soleal and gastrocnemius arteries

no 59


gastrocnemius
Soleus
2 arteries passing through soleus to supply the periphery of gastrocnemius
longitudinal anastomosis on tendo achillis

Experiment (12)

Nos. 58 and 59 (Drawings)

Right external iliac artery of Foetus(D) injected on 1.4.46 with vermilion by hand pressure.

Dissected and Drawn.

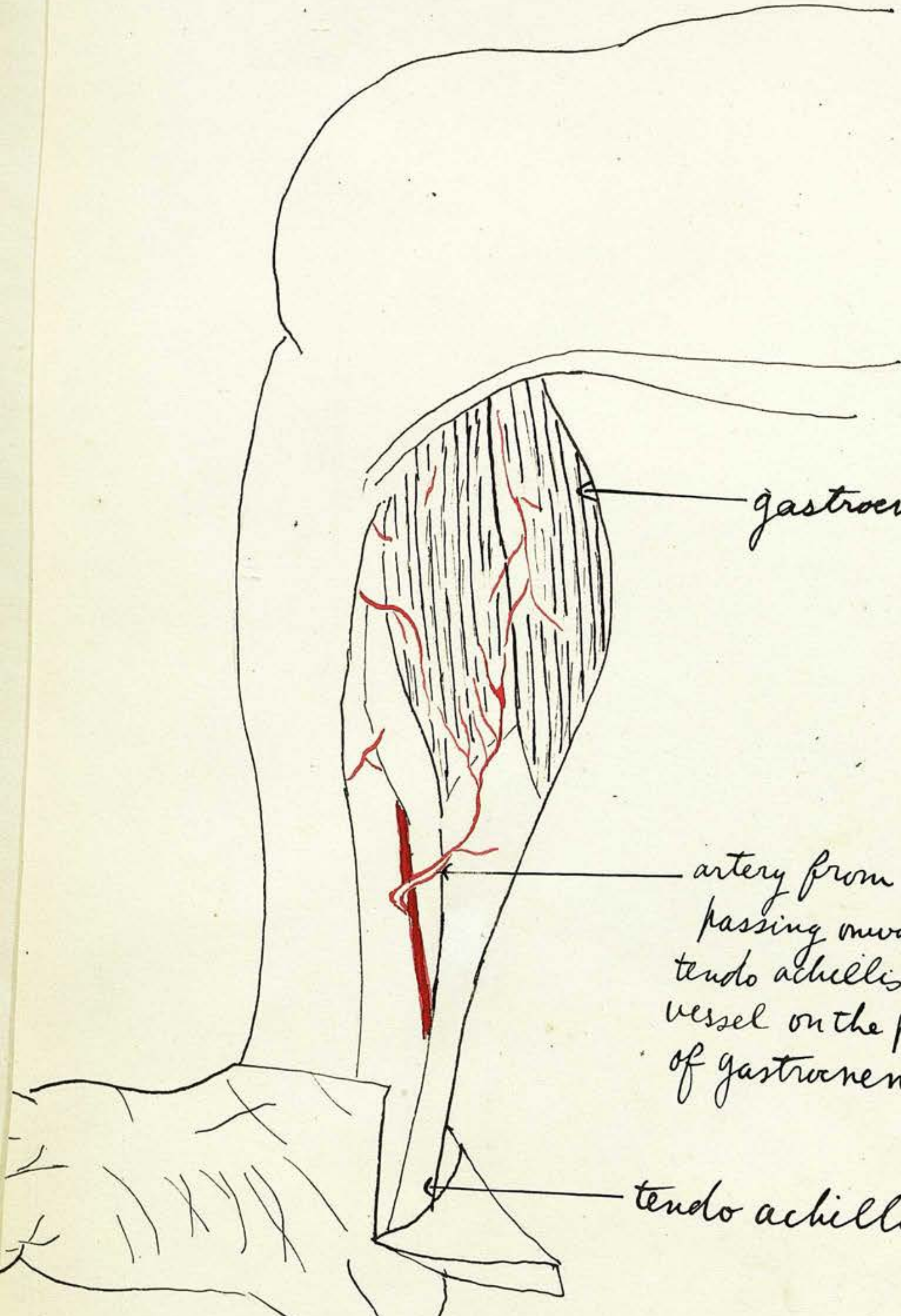
Drawings:-

58 shows a vessel from the posterior tibial penetrating through soleus and anastomosing with one of the arteries of gastrocnemius.

59 shows the arrangement of vessels in soleus, with the longitudinal anastomoses on the tendo achillis, and two arteries passing through soleus to supply the periphery of gastrocnemius.

Experiment (13)/

no 60


gastrocnemius.
artery from posterior tibial
passing onwards after supplying
tendo achillis, to join a slender
vessel on the posterior aspect
of gastrocnemius
tendo achillis

Experiment (13)

No. 60 (Drawing)

Full term male foetus still-born.

Foetus (F)

Specimen - trunk and limbs.

Aorta perfused with water and then injected with starch red lead mixture by hand pressure syringe.

Right leg dissected and drawn.

This shows an artery from the posterior tibial supplying the tendo achillis, and passing onwards to join the slender vessel on the posterior aspect of gastrocnemius, and giving branches to the gastrocnemius muscle as well.

Experiment (14)/

no 61

Photograph of an Xray (lateral view) of a right lower extremity amputated for chronic osteomyelitis. Popliteal artery injected with starch redlead barium sulphate mixture.

Popliteal artery

Branches from popliteal artery going to gastrocnemius

anastomoses between branch of a gastrocnemius artery and a soleal artery

14 141

no 62

Same specimen as no 61, showing arteries of foot and ankle.

3 descending arteries, the anterior, & posterior tibial arteries and the peroneal artery. Small vessels passing posteriorly to supply the tendo achillis.

/

Experiment (14).

Nos. 61 and 62 (Photographs).

Specimen - Adult right lower extremity amputated by Mr. T.E. Rutter at junction of mid and lower thirds of thigh for chronic osteomyelitis of femur.

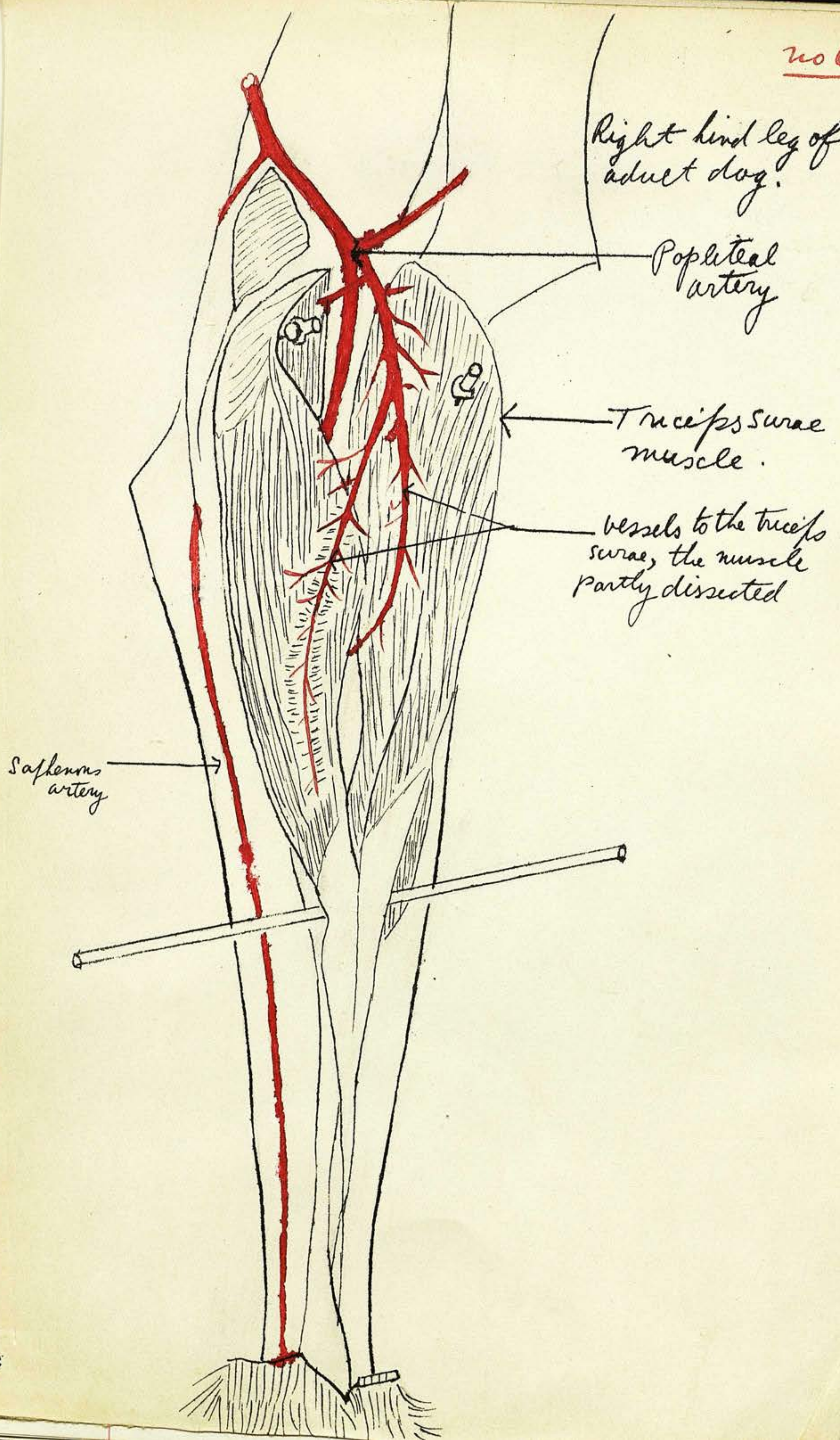
Popliteal artery injected with starch red lead barium sulphate mixture (4:1).

X-rayed and films photographed.

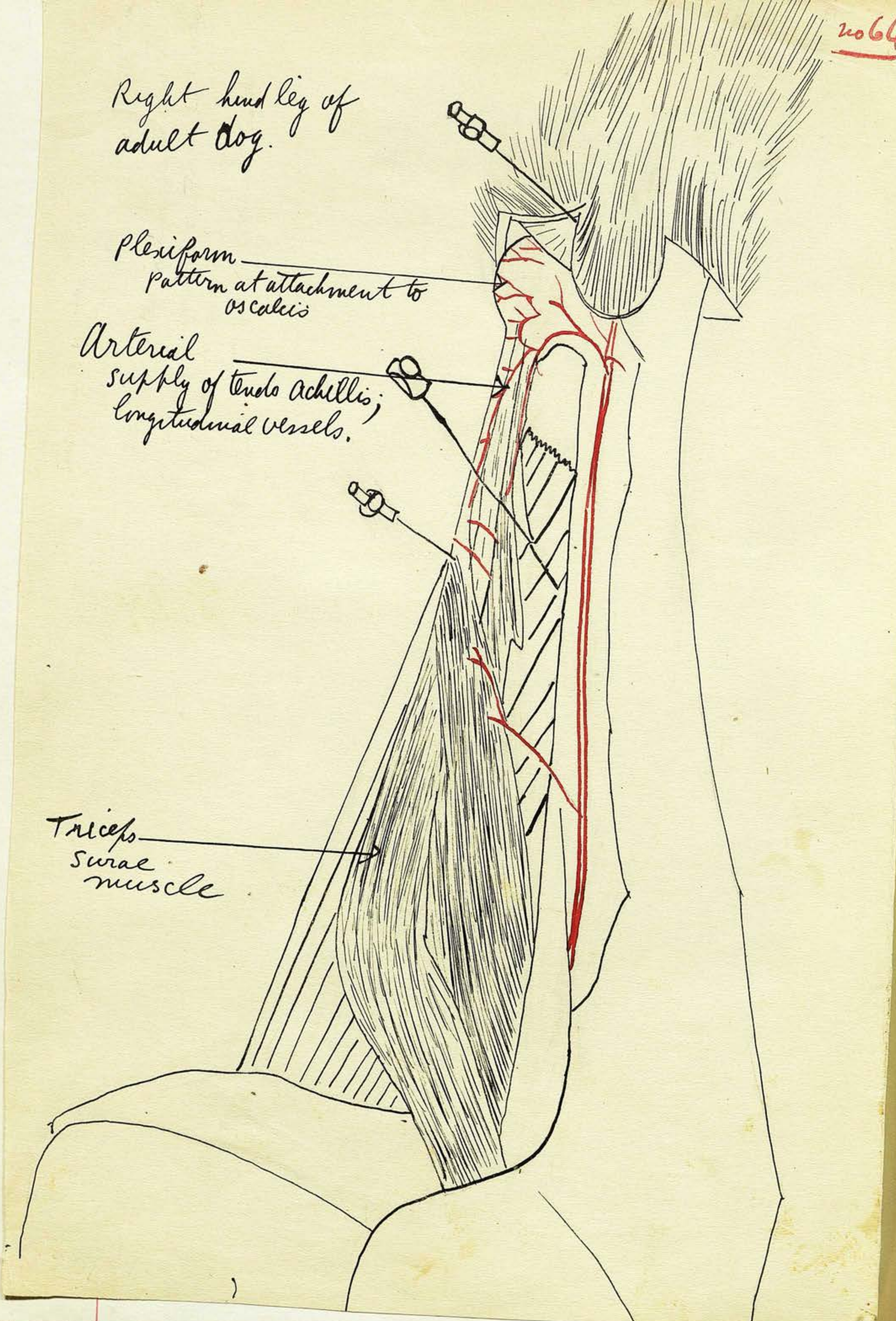
The photographs are reproduced from the X-ray negatives reduced on to a half plate and contact prints taken.

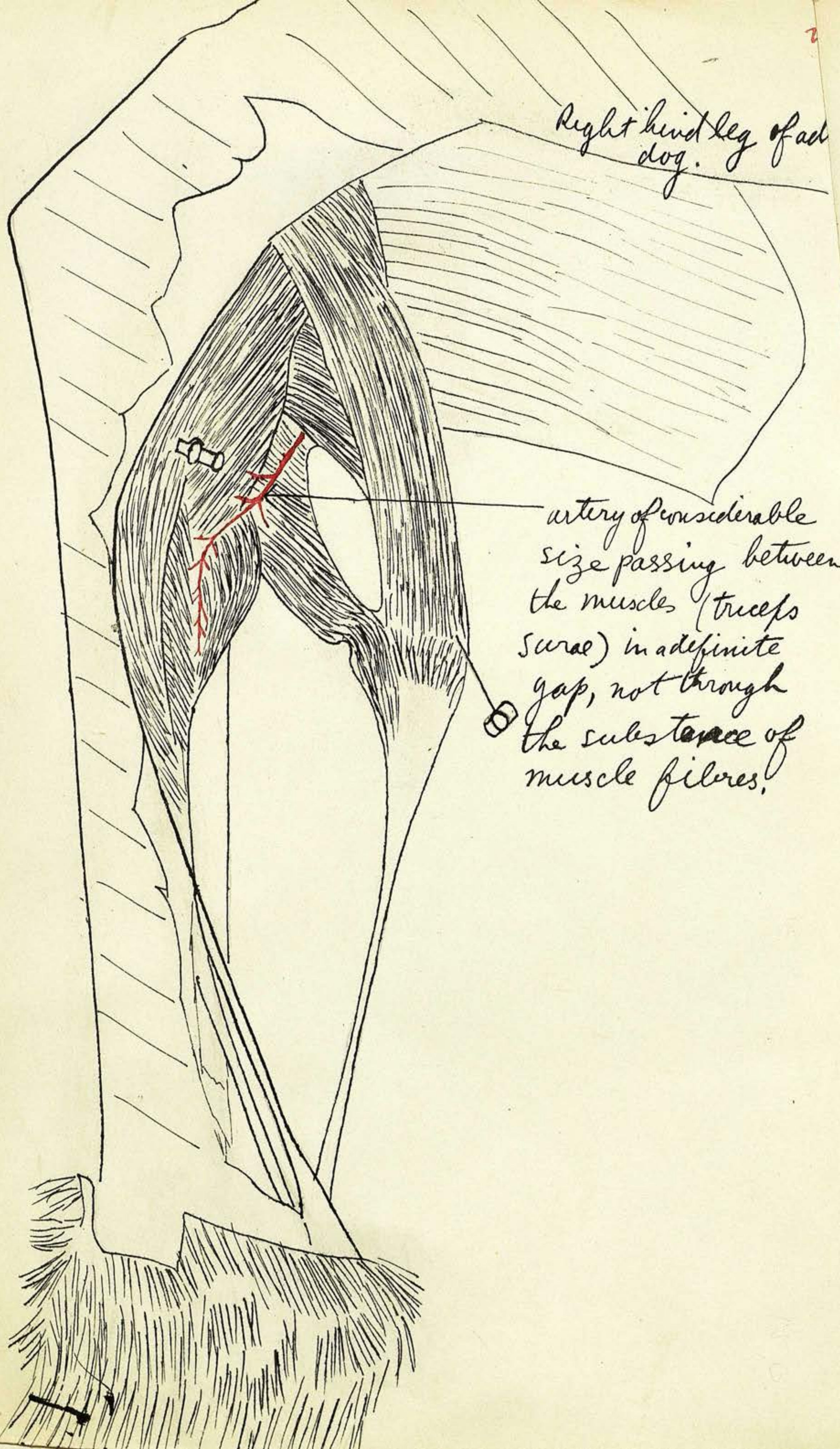
The photographs show the vessels entering gastrocnemius from above, and the soleal vessels entering below. A long slender vessel on the posterior surface of gastrocnemius is seen anastomosing with a branch from one of the soleal arteries. The avascularity of the adult tendo achillis is also well seen. The vessels are as many as in the foetal specimens examined, but in proportion to the size of the adult tendo achillis, are of much smaller calibre.

Experiment (15)/


Right hind leg of aduct dog.
Popliteal artery
Triceps surae muscle.
vessels to the triceps surae, the muscle partly dissected
Saphenous artery

no64


Right hind leg of adult dog.
Plexiform pattern at attachment to os calcis
Arterial supply of tendo achillis; longitudinal vessels.
Triceps surae muscle


Right hind leg of ad dog.
artery of considerable size passing between the muscles (triceps surae) in a definite gap, not through the substance of muscle fibres.

Experiment (15).

Nos. 63, 64 and 65 (Drawings)

Specimen - Right hind leg of adult dog.
(Cadaver from Physiology Department).

Injected with vermilion, then dissected and drawn.

Drawings:-

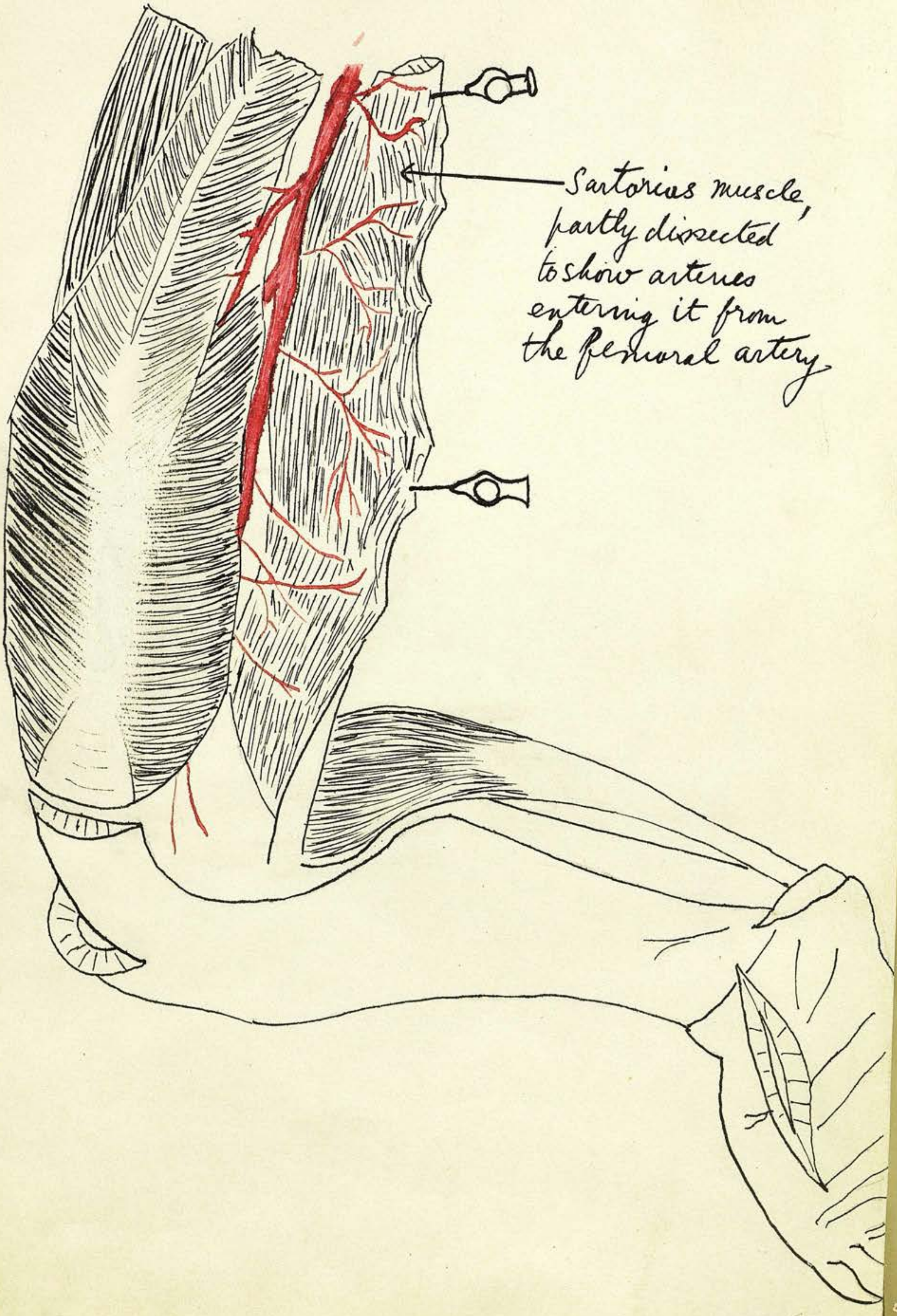
63 shows the triceps surae and main vessels.

64 Shows the arterial supply of the "tendo achillis", the plexiform arrangement of vessels at the attachment to the os calcis and the longitudinal arteries along the tendons.

65 Shows an artery of considerable size passing between the muscles, through a definite interval between them, not through the substance of muscle fibres.

no. 66

Right lower limb of full term foetus.



Experiment (16)

No. 66 (Drawing)

Specimen - Right sartorius of Foetus (A).

Dissected.

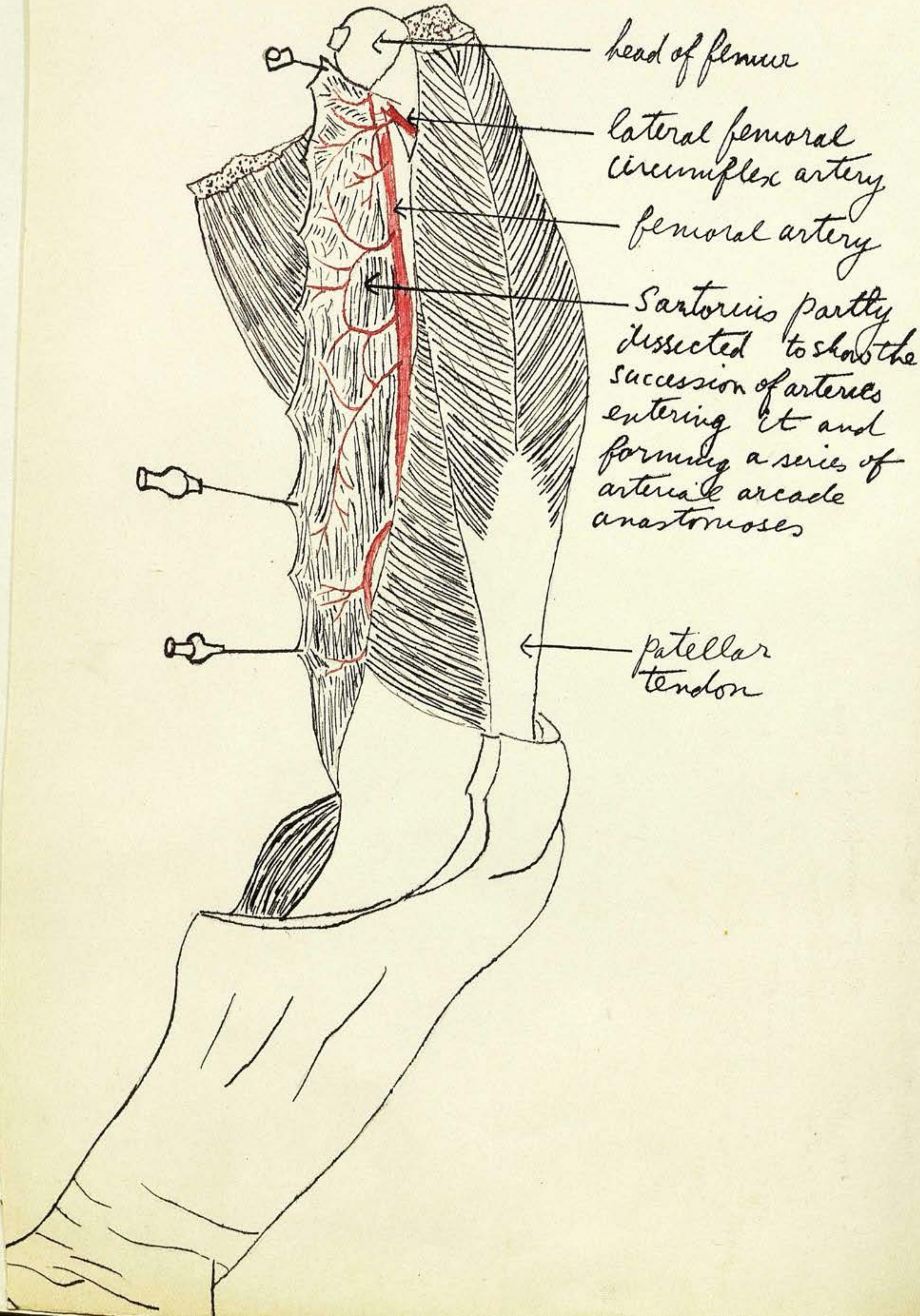
Note the regular succession of arteries entering the muscle from the femoral, and the descending genicular arteries branching into fine longitudinal channels within the substance of the muscle.

No anastomoses demonstrated in this specimen.

Experiment (17)/

no 67

Left lower limb of full term foetus



Experiment (17).

No. 67 (Drawing)

Specimen - Left sartorius of Foetus (B).

Dissected.

Note the succession of vessels passing from:-

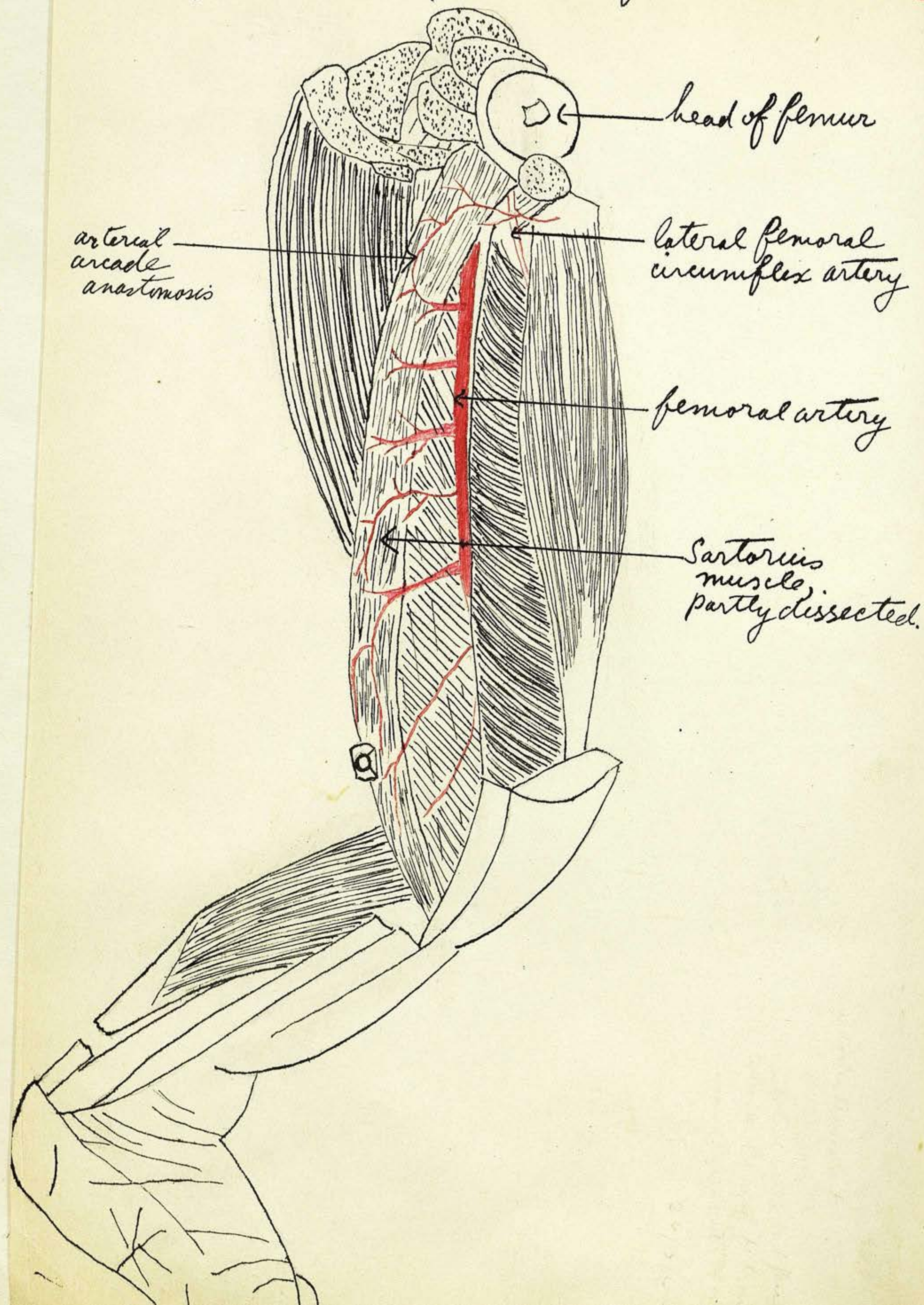
1. The lateral femoral circumflex.
2. The femoral.
3. The descending genicular arteries, to the muscle, and forming a series of arterial arcade anastomoses between themselves in the substance of the muscle.

   The finer ramifications run parallel with the muscle fibres.

Experiment (18)/

Left lower limb of full term foetus

2068



Experiment (18).

No. 68 (Drawing)

Specimen - Left sartorius of Foetus (C).

Dissected.

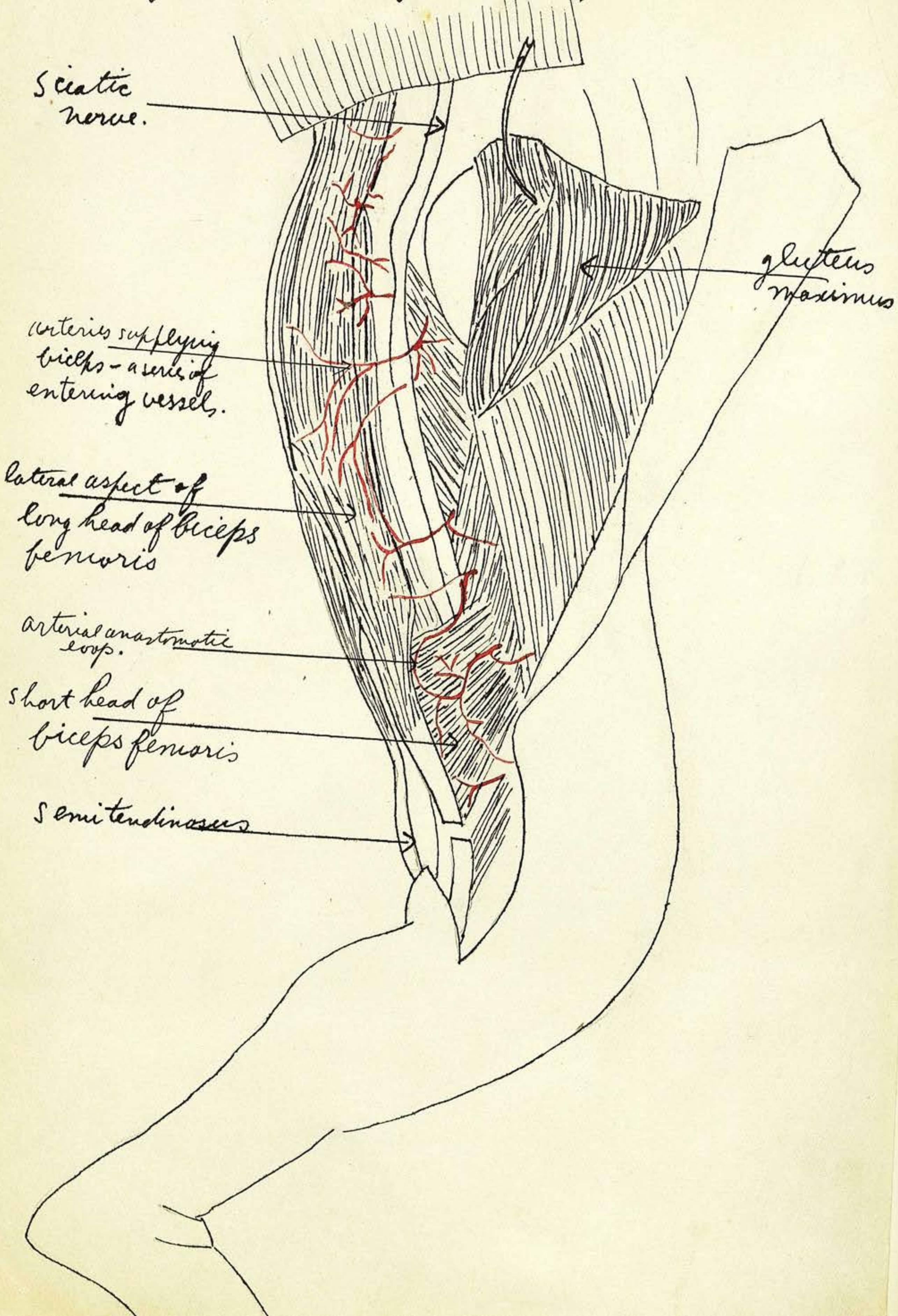
Note the arterial arcade anastomoses between the branch from the lateral femoral circumflex artery and the uppermost branch from the main femoral.

Same findings noted as in (17).

Experiment (19)/

no 69

Right lower limb of full term foetus



Experiment (19).

No. 69 (Drawing) and No. 70 (Drawing).

Specimen - Right biceps femoris, Foetus (D).

Dissected.

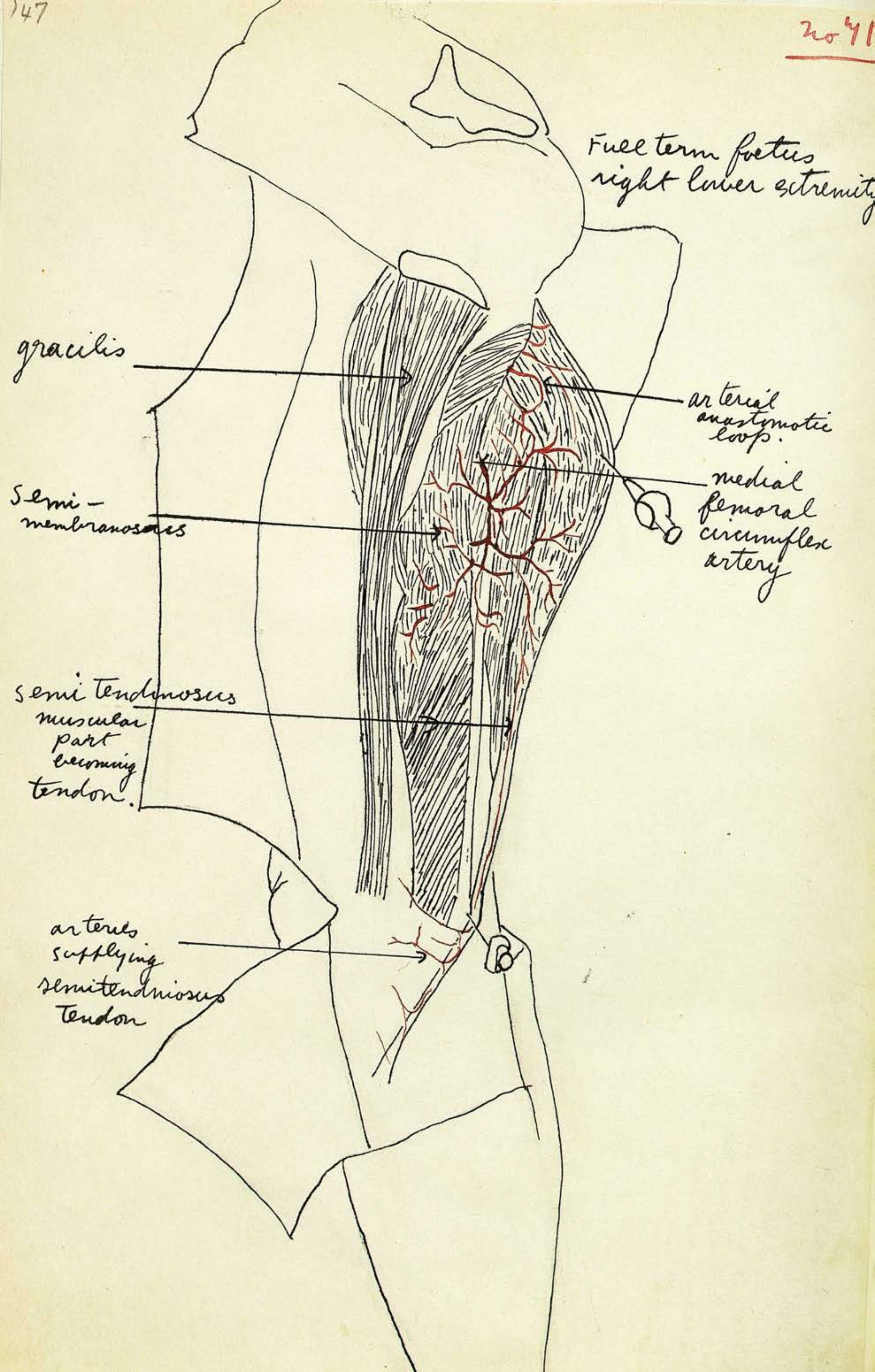
Drawings:-

69 Lateral aspect.

70 Medial aspect.

These show the blood supply as a series of vessels entering the two heads from above downwards. Those entering the short head are parallel with the fleshy fibres arising from the bone, and form anastomotic loops in the substance of the muscle.

Those entering the long head take the same direction as the muscle fibres after entry.


Full term foetus
right lower extremity
gracilis
arterial anastomotic loop.
Semi-membranosus
medial femoral circumflex artery
semi tendinosus
muscular part becoming tendon.
arteries supplying semitendinosus tendon

Experiment (20)

No. 71 (Drawing)

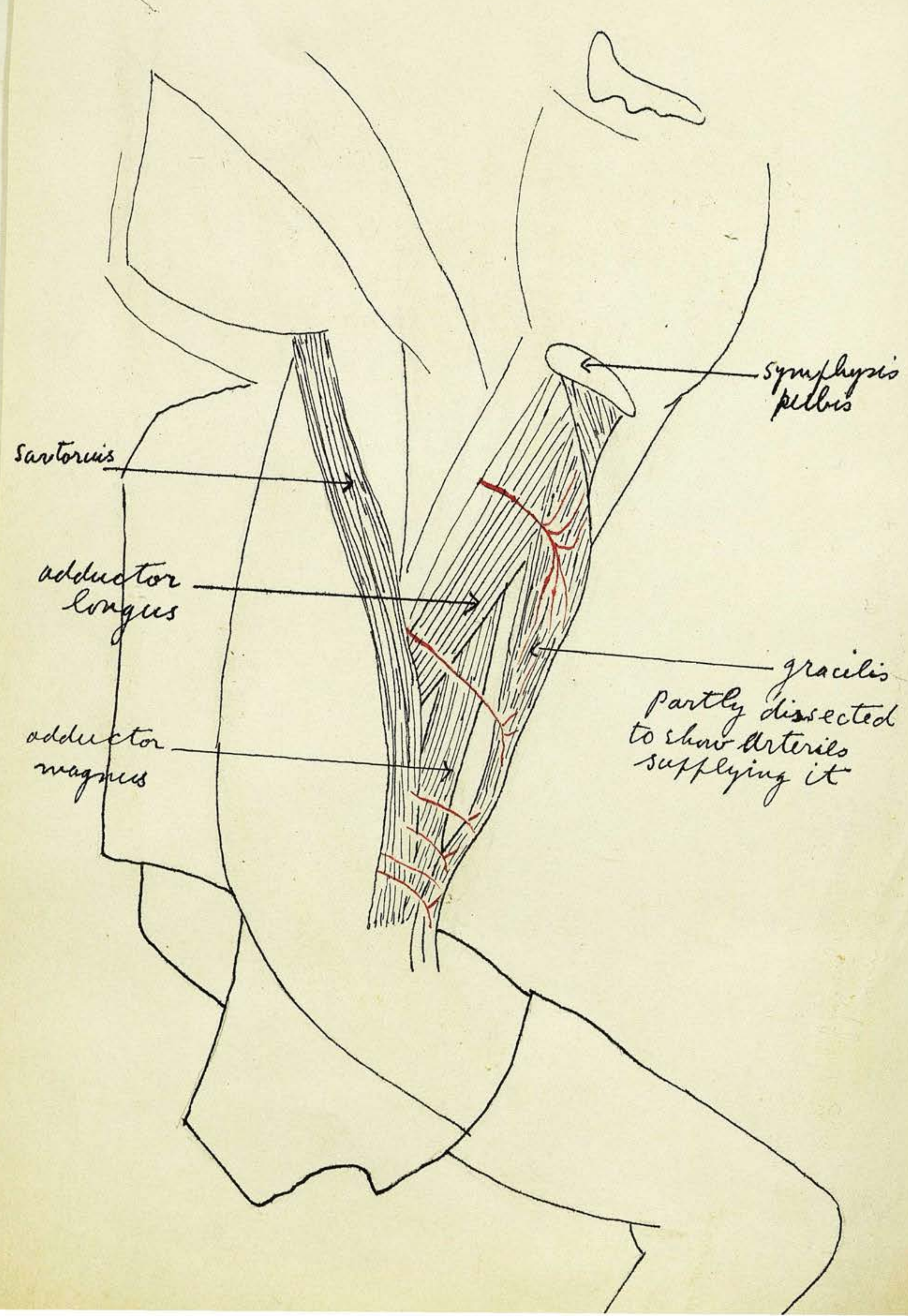
Specimen - Right semitendinosus. Foetus (D).
Dissected.

The drawing shows the main vessels entering the belly of the muscle, arising from the medial femoral circumflex; two anastomotic loops are noted at the superior end of the muscle. A very long slender vessel runs along the tendon which is also supplied near its insertion by further vessels coming in to join the longitudinal channel.

Experiment (21)/

no 72

Full term foetus right lower extremity



Experiment (21).

No. 72 (Drawing).

Specimen - Right gracilis. Foetus (D).

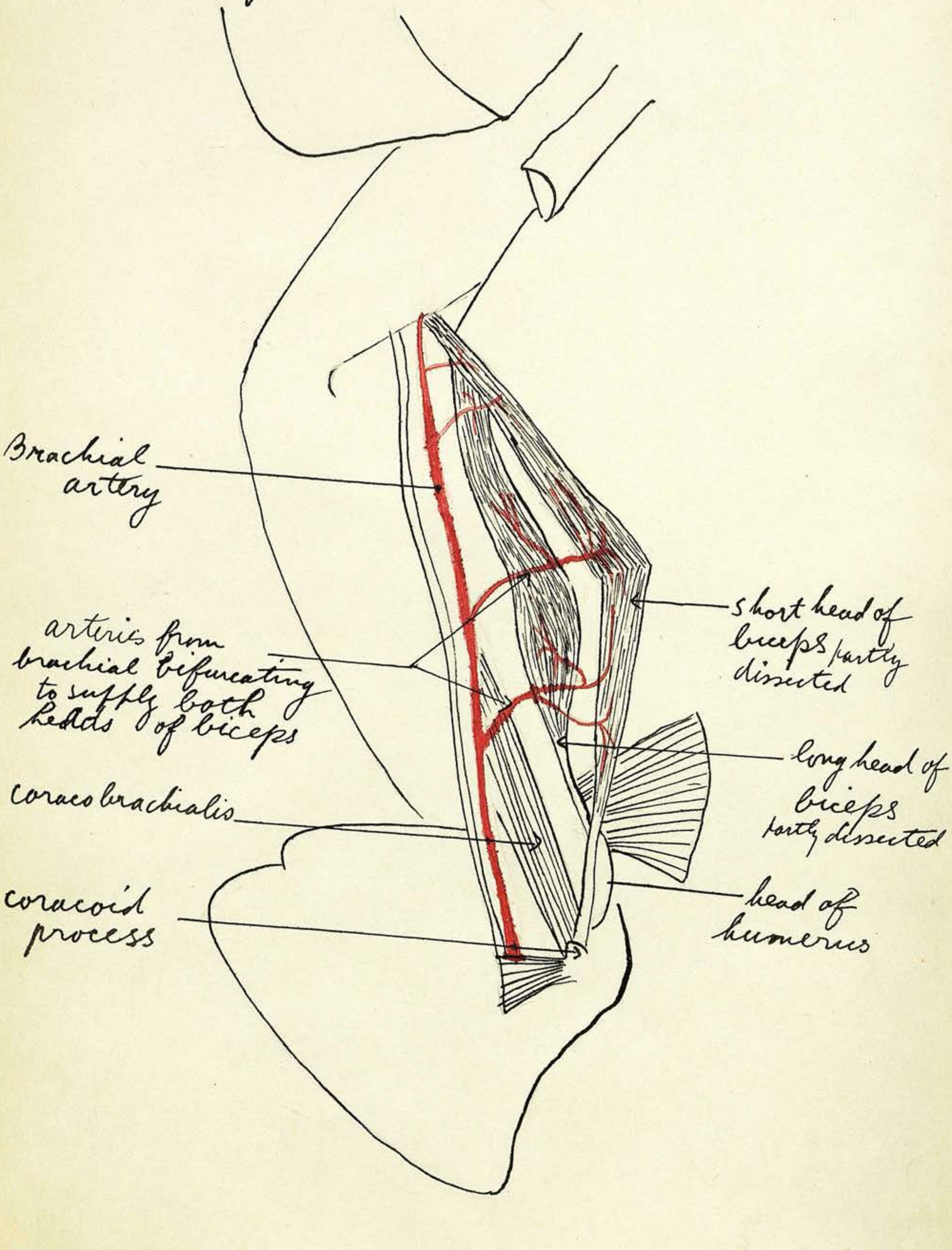
Dissected.

One main vessel from the medial femoral circumflex the main trunk dividing into longitudinal channels in the substance of the muscle.

Several slender vessels enter lower down, one from the main femoral, the others from the descending genicular artery, supplying the tendon.

no 73.

Right upper extremity of full-term foetus



Experiment (22)

No. 73 (Drawing).

Specimen - Right biceps brachii. Foetus (D).

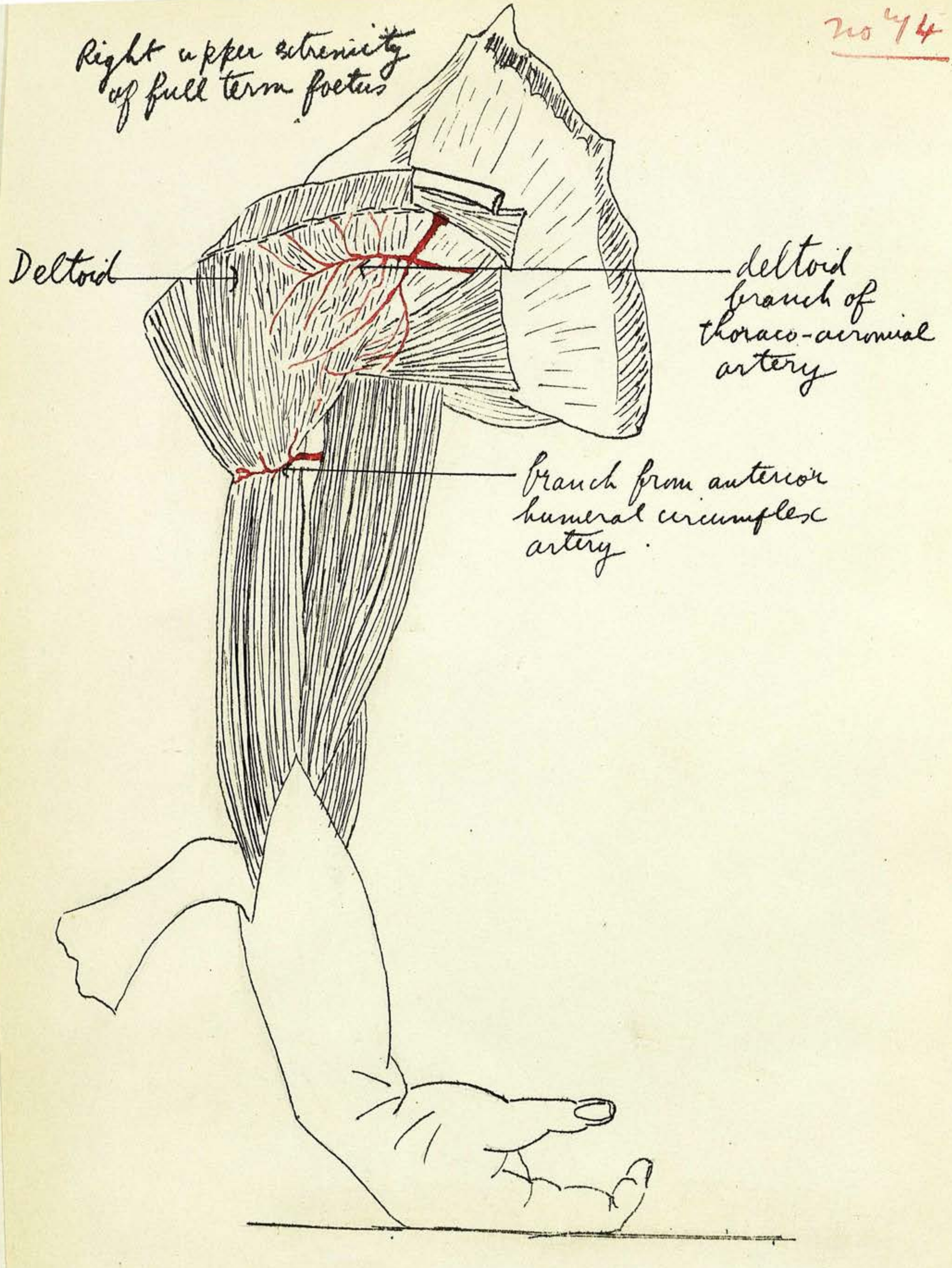
Dissected.

This shows several arteries passing from the brachial bifurcating into separate branches to supply the long and short heads respectively.

Two main arteries were seen; accompanying the upper one was the branch to the biceps from the musculo cutaneous nerve, i.e. a definite neurovascular bundle entered a hilum. The nerve is not shown in this drawing. The artery distal to this was of equal size.

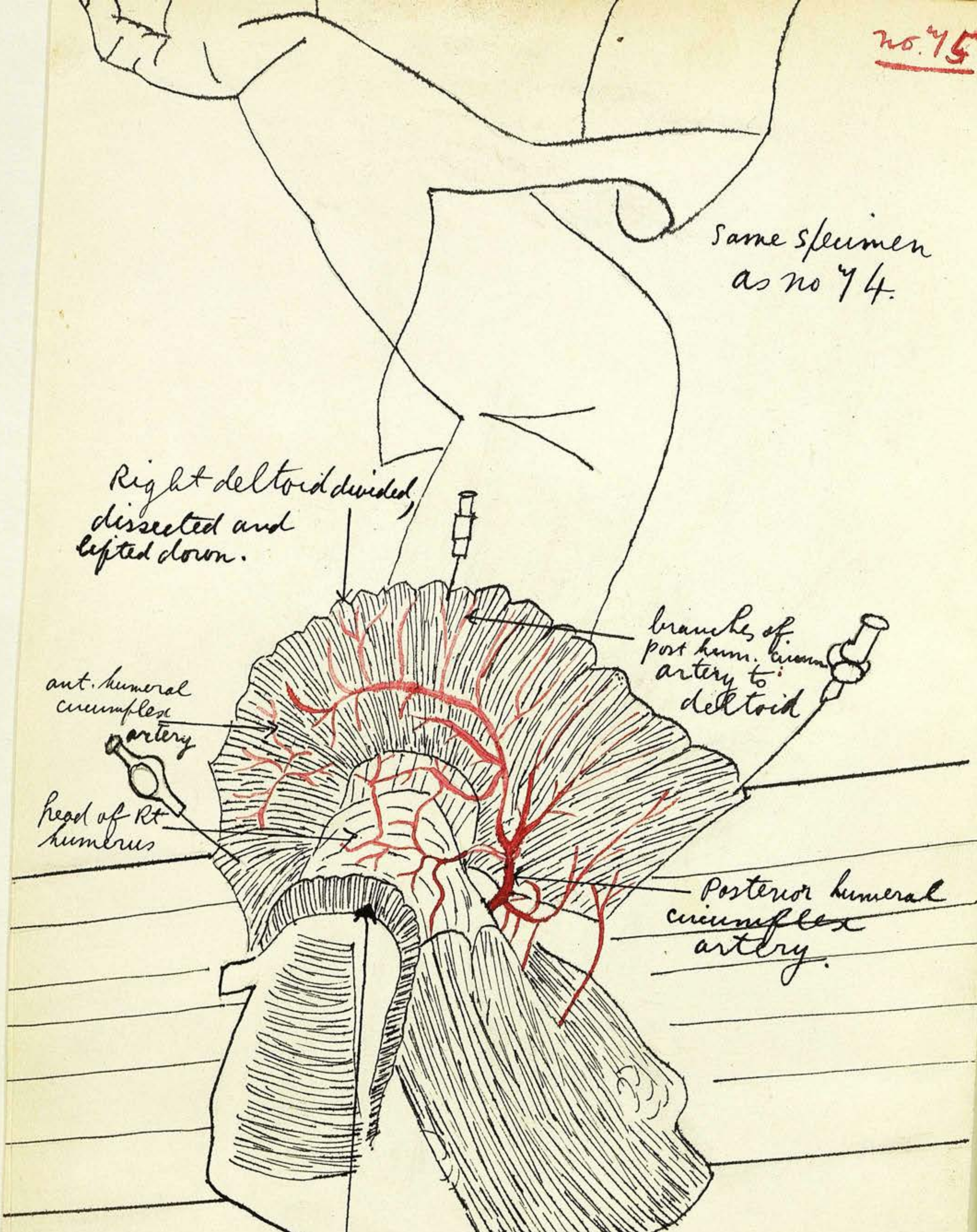
Experiment (23)/

No 74


Right upper extremity of full term foetus
Deltoid
deltoid branch of thoraco-acromial artery
branch from anterior humeral circumflex artery.

no. 75

Same specimen as no 74.



origin of deltoid from clavicle and Scapula.

Experiment (23).

Nos. 74 and 75 (Drawings)

Subclavian artery of Foetus (D) injected with vermilion 1.4.46.

Right deltoid dissected.

The arterial pattern is demonstrated in the two drawings.

(a) A circular main vessel, the posterior humeral circumflex, with a series of single vessels coming off it running in the grooves between the muscle fasciculi, and dividing into finer vessels in the substance of the muscle.

(b) A continuation in the anterior portion of the same pattern by the anterior humeral circumflex, which does not anastomose with the posterior humeral circumflex.

(c) Branches at the periphery.

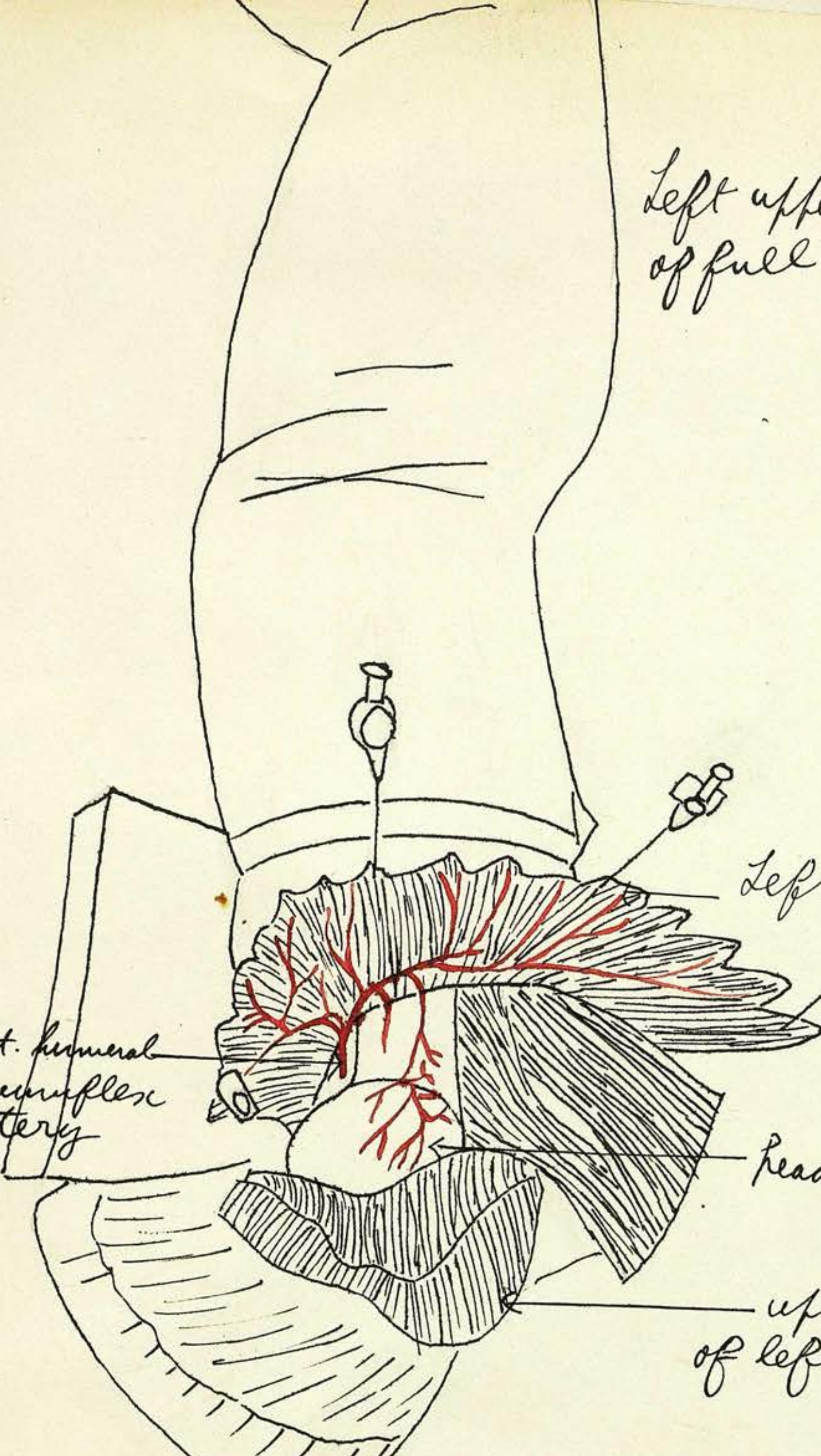
i. anteriorly from the deltoid branch of the thoraco acromial artery.

ii. posteriorly from the circumflex scapular.

iii. at its insertion, from the anterior humeral circumflex artery.

no 4

Left upper extremity of full term foetus



Experiment (24)

No. 76 (Drawing).

Female foetus died 10.4.46 of icterus haemorrhagica neonatorum. Foetus (E).

Left subclavian artery injected with vermilion with hand pressure syringe.

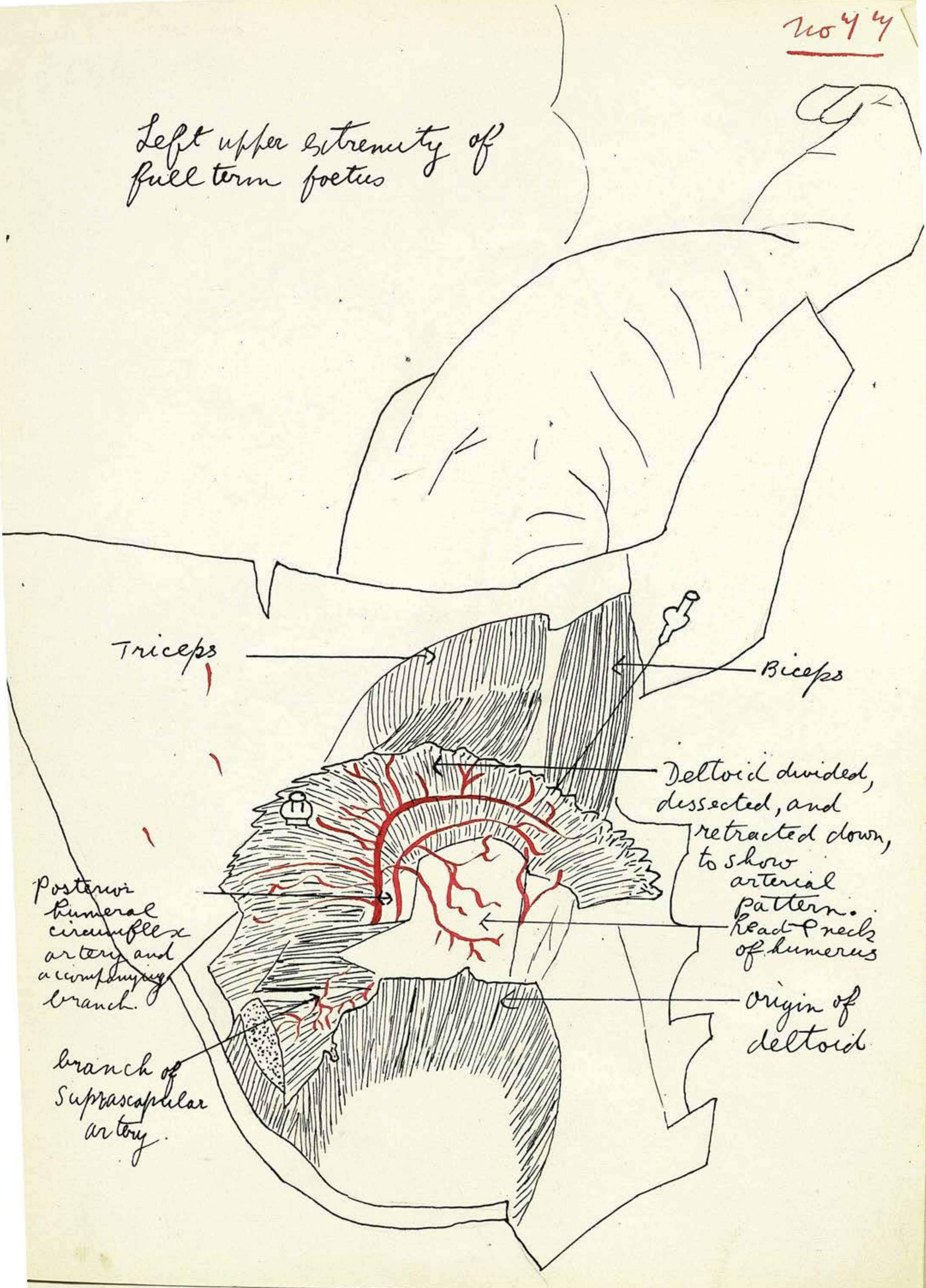
Deltoid dissected.

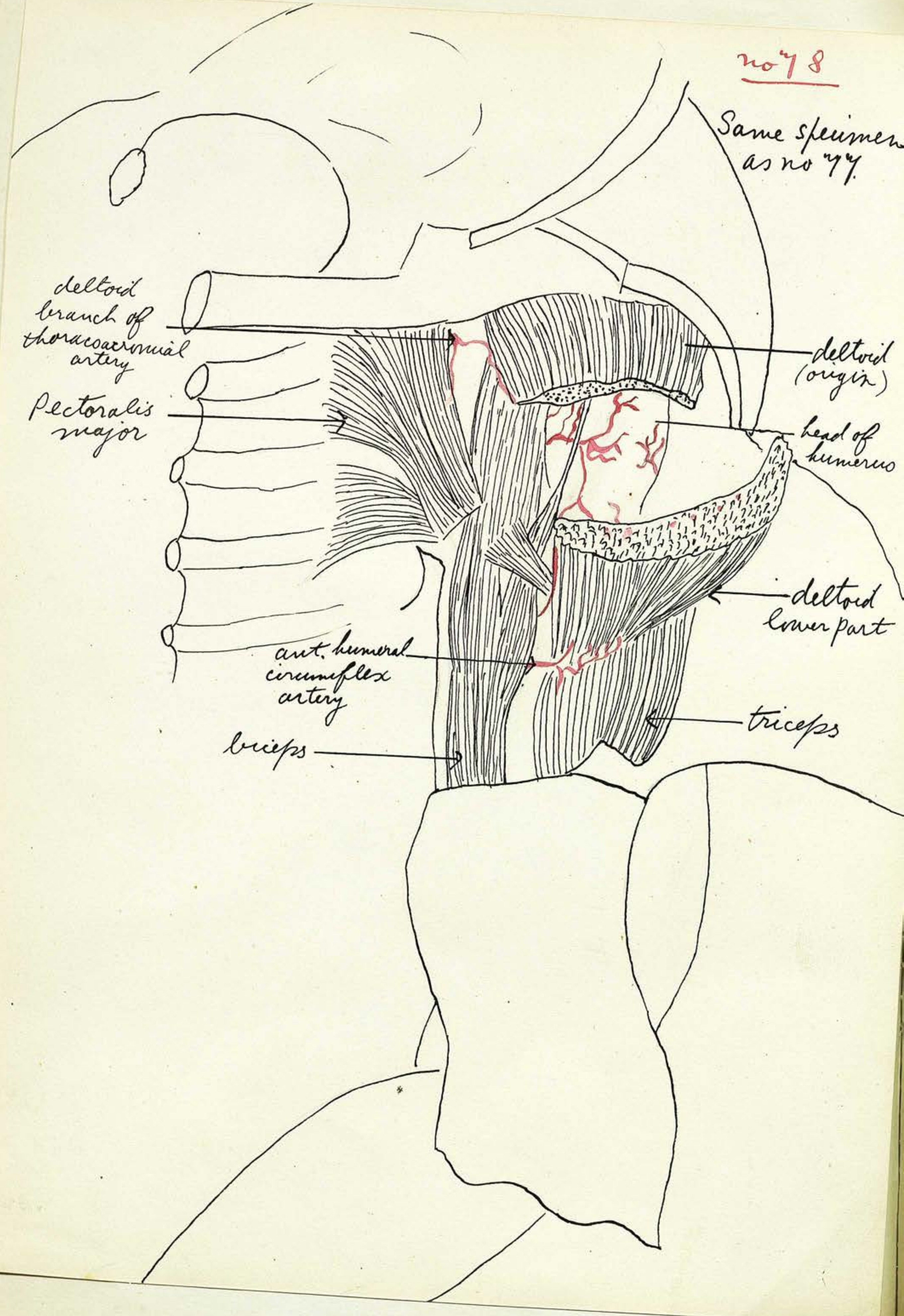
This specimen shows the circular posterior humeral circumflex trunk giving off straight vessels to ramify at different depths in the substance of the deltoid muscle.

Experiment (25)/

No 44

Left upper extremity of full term foetus




no 78
Same specimen as no 77.
deltoid branch of thoracoacromial artery
Pectoralis major
deltoid (origin)
head of humerus
deltoid lower part
ant. humeral circumflex artery
triceps
biceps

Experiment (25).

Nos. 77 and 78 (Drawings)

Left subclavian of Foetus (F) injected with starch and red lead with hand pressure syringe.

Left deltoid dissected.

This shows, in drawings 77 and 78, a double main circular channel formed by the posterior humeral circumflex and a branch, with vessels passing off into the muscle substance. The anterior portion received a branch from the anterior humeral circumflex artery.

The periphery is supplied from:-

(a) the thoraco-acromial artery

(b) the anterior humeral circumflex artery

(c) the suprascapular artery.

Experiment (26)/

3

no 79

Corrosion cast of vessels of left upper extremity of full term foetus.

vessels too numerous to show details required. See text.

Experiment (26)

No. 79 (Photograph).

Left subclavian artery of Foetus (D) injected with celloidin.

Satisfactory cast obtained.

Photographed.

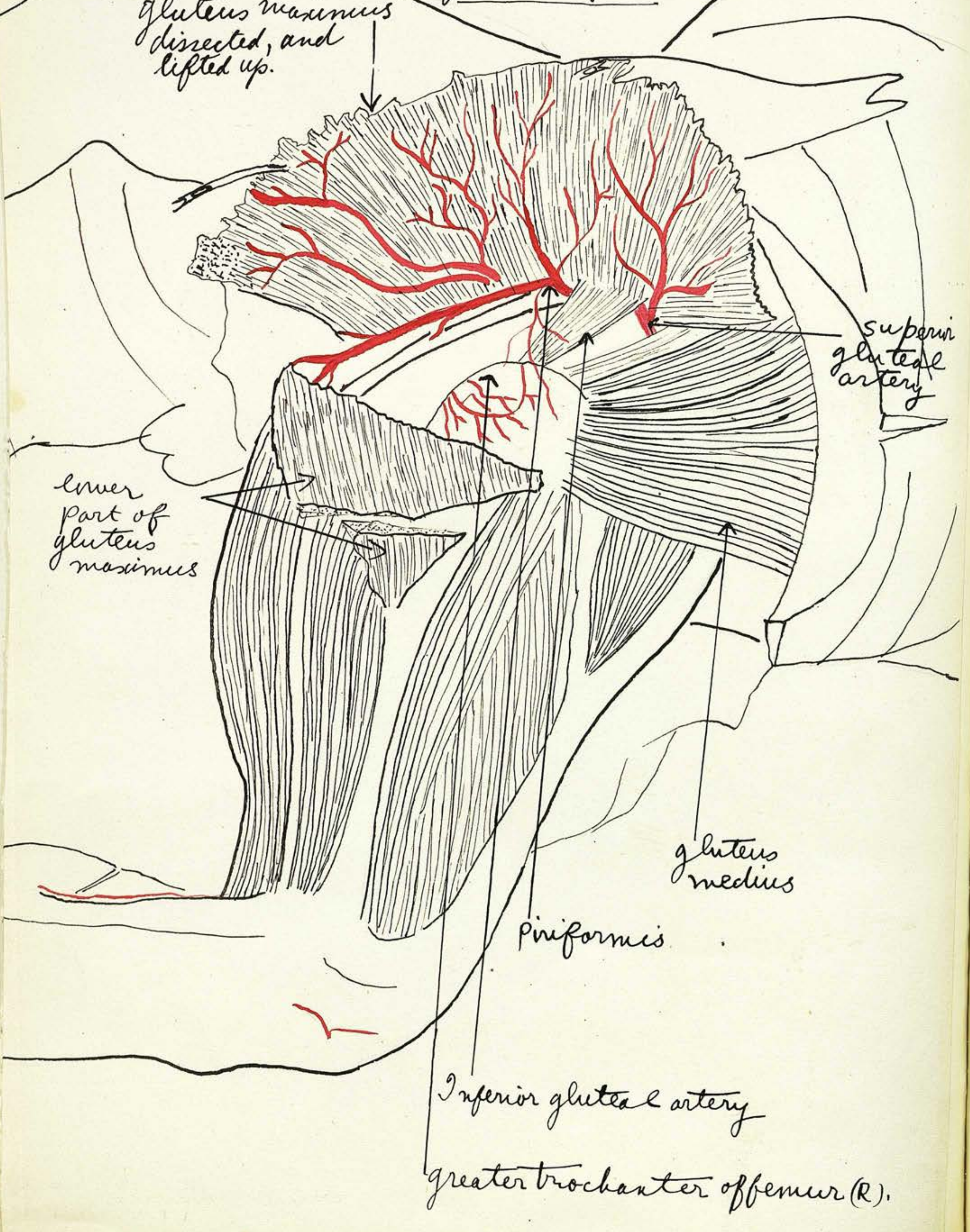
The vessels are too numerous to show well on the photograph, the result therefore not being as satisfactory as the dissection method.

This cast showed a slender anastomotic vessel between the anterior and posterior humeral circumflex arteries.

Experiment (27)/

Rt lower extremity of full term foetus

no 80



Experiment (27)

No. 80 (Drawing).

Right gluteus maximus of Foetus (F), injected with starch and red lead.

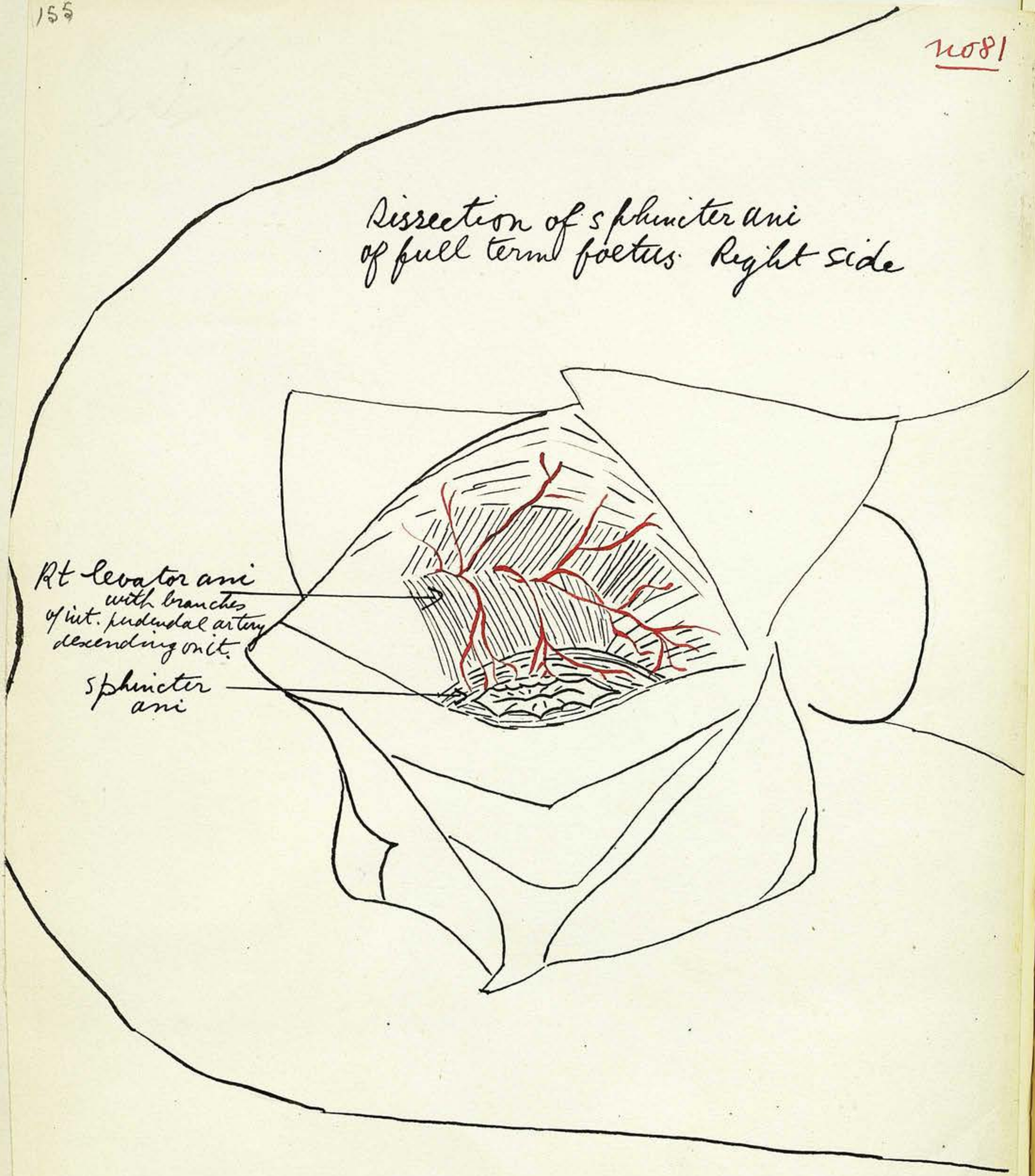
This specimen shows the vessels entering the muscle from the superior and inferior gluteal arteries, running parallel with the muscle fibres.

No anastomoses between the superior and inferior gluteal arteries seen.

Experiment (28)/

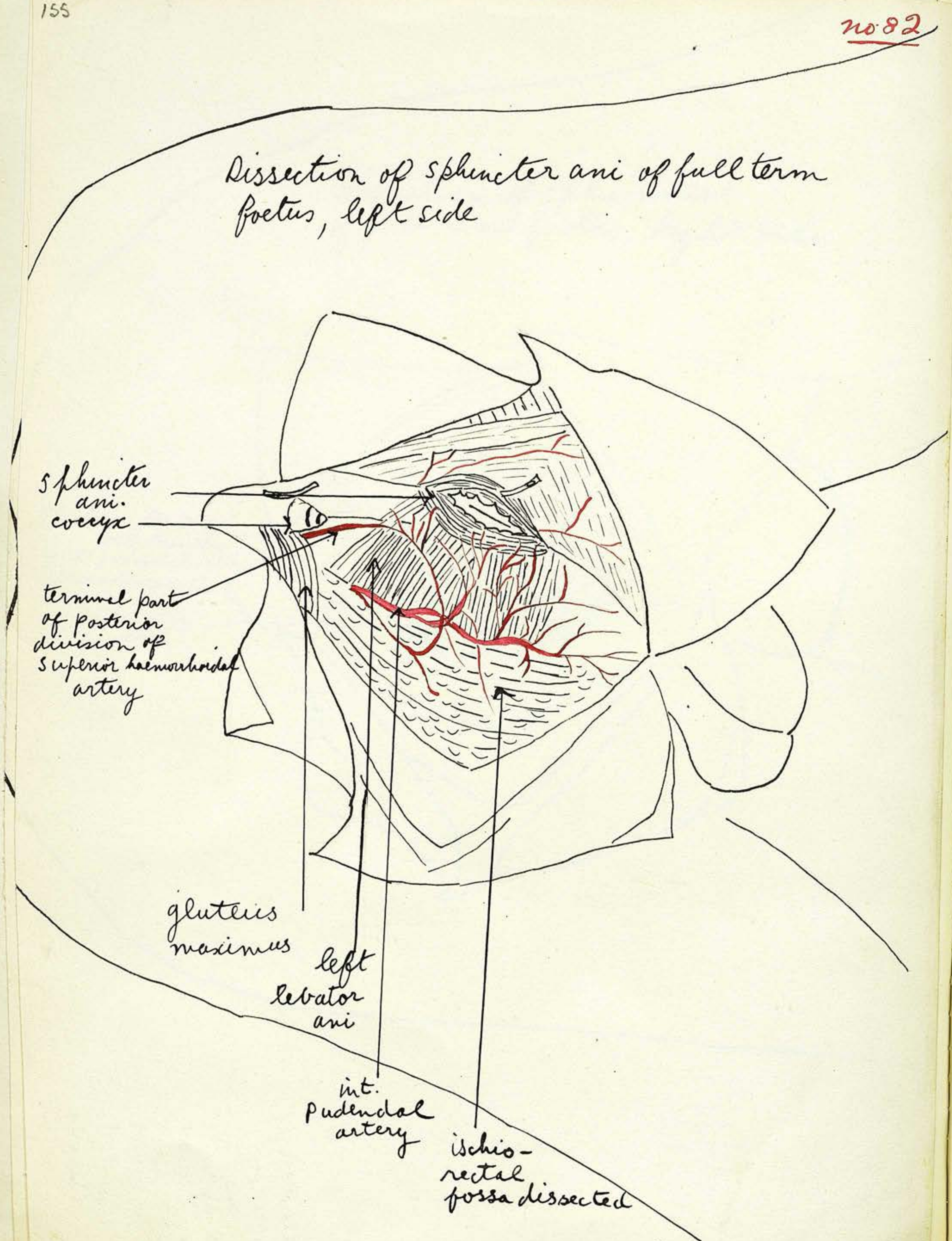
no81

Dissection of sphincter ani
of full term foetus. Right side



no. 82

Dissection of sphincter ani of full term foetus, left side



Experiment (28).

Nos. 81 and 82 (Drawings).

Dissection of the sphincter ani of Foetus (F).

Drawings 81 and 82 show the right and left sides of the levator ani and external sphincter muscles.

Note the arteries descending from the internal pudendal, on the levator ani, to the external sphincter. On the left side there is an arcade anastomosis with an artery descending near the posterior border of levator ani. This artery, on further dissection, was found to be the continuation of the posterior division of the superior haemorrhoidal artery.

From/

CONCLUSIONS.

In these experiments it is seen that the arrangements of the arterial pattern in skeletal muscle is well adapted to serve the peculiar features of this particular structure, especially as regards its contractile nature.

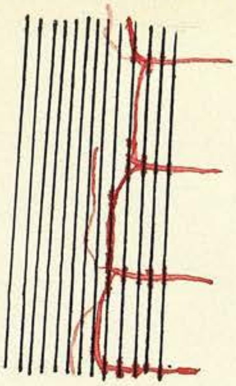
It can be stated that muscles receive a main blood supply, and a minor supply. The main blood supply is the supply to the larger part of the muscle substance, and differs according to the different type of muscles. The minor supply is the series of vessels which supply the edges and borders of the muscle, and the insertions of its tendon. These vessels are small and have only sparse arterial anastomosis with the main vessels.

In/

Vascular patterns in Skeletal muscle.

no 83

1.

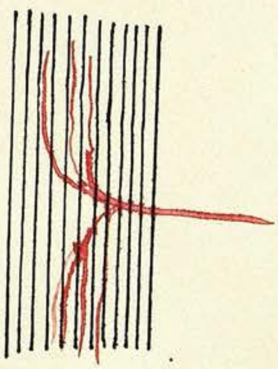


a series of separate vessels entering the muscle throughout its length, joined together by a longitudinal anastomotic channel. eg sartorius.

2.

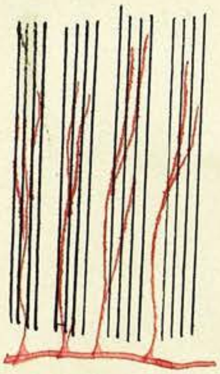
a longitudinal pattern of vessels derived from a single group of arteries which arises from vessels entering one end of the muscle, eg. gastrocnemius.

3.



Radiating pattern of collaterals arising from a single main vessel entering a hilum about its middle eg gracilis.

4.



a series of separate vessels entering the muscle with very few anastomoses between them, eg deltoid.

No. 83 (Drawing)

In the muscles studied in the above experiments, the vascular patterns of the main blood supply were of four varieties:

1. A series of separate vessels entering the muscle throughout its length, joined together by a longitudinal anastomotic channel formed in some places as a series of anastomotic loops. This corresponds to (1) and (4) of Blomfield's classification (1945), the division into two groups on account of the straightness of the curve of the anastomotic channel being considered perhaps an unnecessary elaboration. This pattern is present in the quadrilateral and fusiform muscles, e.g. sartorius, biceps femoris.

2. A longitudinal pattern of vessels derived from a single group of arteries which arises from vessels entering one end of the muscle. e.g. gastrocnemius.

3. Radiating pattern of collaterals arising from a single main vessel entering a hilum about the middle, with sparse anastomotic connections with smaller vessels entering nearer the ends, e.g. gracilis.

4. A series of separate nutrient arteries entering the muscle with very few anastomoses between them, e.g. deltoid, gluteus maximus.

It is generally accepted that the biceps brachii is a muscle whose blood supply is derived from a single main vessel entering it along with its supplying nerve, the neurovascular bundle passing in through a well defined hilum. This is the pattern constantly seen in the adult. The/

The case described by Wood Power (1945) already previously mentioned, in which complete gangrene of the long head of biceps developed following the severing of the single artery entering it, was an example of this pattern. The specimen illustrated in the present series of experiments, showed two main arteries coming off the brachial artery, equal in size, and each dividing into branches, one for the long and one for the short head of biceps. Another foetal limb examined showed the same appearance. It is probable that the pattern at birth may differ from that in the adult, one vessel continuing to develop along with the muscle, others, of equal size at birth remaining stationary and appearing in the adult as of inconsiderable size. One main vessel is certainly more in keeping with the function of the adult biceps which has such a range of movement over the deeper tissues, than a series of vessels, the lower ones of which would be greatly stretched during active contraction and act as mooring ropes checking the muscle's action.

The whole basis underlying vascular patterns in muscle is the adaptation of the vessels to the contraction and movement of the muscle.

Gastrocnemius/

Gastrocnemius lies superficial to soleus deep to which are two big arteries, the posterior tibial and the peroneal. From these main trunks vessels arise and sink into soleus; but they do not extend through soleus, a contractile element, into gastrocnemius. If the main blood supply of gastrocnemius was derived from arteries passing through soleus, then every time the soleus contracted the gastrocnemius would become ischaemic, by temporary cutting off of its blood supply. To prevent this, the arteries to gastrocnemius arise direct off the popliteal, and enter the muscle superficial to soleus. Only a few peripheral arteries pass through the lateral border of soleus into gastrocnemius (see muscle experiments (11) and (12)).

This tendency of the course of a main vessel to avoid passing through another muscle first is also seen in semitendinosus lying superficial to semimembranosus, deriving a blood supply from the medial femoral circumflex artery, by vessels passing to it clear of the semimembranosus. It was also well seen in the muscles of the dog examined (Drawing 65). Vessels going through one muscle to another appear therefore to be rare.

The/

The relation of muscular vessels to the muscle's contraction is also seen in the loop formation present in the contractile portion, whereas the non contractile portion, the tendon, has long straight vessels. An example of this is seen in the gracilis (muscle experiment 21), and in all the specimens of triceps surae and tendo achillis examined. A further illustration is seen in the arterial pattern of the deltoid, which is beautifully adapted to the contraction of that muscle. This pattern can be compared with the blood supply of the colon. The posterior humeral circumflex artery corresponds to the marginal artery, and the single branches coming off and running parallel with the muscle fibres may be compared with the vasa recta in relation to the circular muscle of the gut.

In the better injected specimens fine arterial anastomoses became evident which were not apparent in others, and first impressions from earlier experiments required correction. Slender anastomotic channels at the periphery, the "minor" supply were noted between soleus and gastrocnemius (muscle experiments(11) and (12)) and in the deltoid between posterior and anterior humeral circumflex arteries (muscle experiment (26)).

These/

These anastomising vessels play their part in re-establishing circulation in a muscle, when the main vessel is obstructed. It was noted in Le Gros Clark's experiments that the revascularisation of the muscle in rabbits after ligation of the main vessel took some days, becoming more complete the longer the interval between ligation and examination. During this interval, the muscle involved was in a precarious state of viability, till the new circulation asserted itself. This precarious state of viability following interference with the main blood supply, the causal agent being a lesion of an artery in a wound, was found to be a most important predisposing factor in anaerobic infections, in the War.

In certain instances also, it was noted that division of an artery in a wound had led to complete gangrene of the muscle it supplied, (Wood Power 1945). Arteries of muscle, in such cases, act therefore to all intents and purposes as end arteries. Gangrene and infection by gas gangrene organisms follow their division.

In/

In war wounds, however, there is a complicating factor not seen with experimental animals. The passage of a projectile causes destruction to a greater or lesser extent beyond the actual wound track, and the other vessels which constitute the minor supply of a muscle may be involved by actual division, or by contusion. Thus fine channels which would dilate in the experimental animal, and produce eventually an adequate new circulation, are not available in a war wound, and a bigger and more permanent area of local ischaemia of a muscle results up to the level of the next main vessel entering. Such muscles therefore as gastrocnemius or gracilis are placed in jeopardy when their entering vessels are damaged, whereas soleus or sartorius stand a better chance. The free excision of damaged areas and the careful conservation of vessels entering muscle, advocated in the treatment of war wounds, is therefore seen to have a sound anatomical basis.

SUMMARY/

SUMMARY.

A preliminary discussion about the course of arteries with particular reference to anastomoses and end-arteries, and with some observations based on Hilton's work, introduced this investigation. Some of the literature was briefly reviewed, and the different views by different authorities, especially in regard to the vessels of the intestine were discussed.

The arteries of certain regions were then selected for study with a view to seeing if there was any particular association of the form of the vessels with the function of the organ they supplied. For that reason the arteries of
1. The placenta, 2. the pulmonary bronchial arteries, 3. the intestinal arteries,
4. the arteries of skeletal muscle were studied.

Various methods of demonstrating the course of arteries were employed;

1. The celloidin corrosion cast technique, of which a description is given. It was found to be satisfactory for the placenta, an ideal organ for injection as, owing to the arrangement of vessels as a series of end arteries, the leakage is minimal and hence a good cast assured./

assured. The use of different coloured celloidins, stained red for one umbilical artery, stained blue for the other, was satisfactory in determining if any peripheral anastomoses were present between the two arteries.

The celloidin cast technique, using two different coloured celloidin solutions was equally satisfactory in determining if there were any macroscopic junctions between the pulmonary and the bronchial arteries.

The celloidin corrosion technique was particularly suitable in specimens of the intestine, as these thin tissues were easily corroded in acid, and the casts leant themselves readily to photography. It was not so satisfactory in specimens of skeletal muscle especially in the foetus; once the soft tissues were corroded it was difficult to estimate where exactly the vessels had been situated, even with the corresponding limb of the opposite side injected with starch and red lead and dissected as a control.

2. Injection with starch and red lead and dissection proved a useful method for demonstrating the course of arteries in skeletal muscle.

3. Injection with a preparation of vermilion and then X-raying was not very successful either in the placenta or in skeletal muscle, owing perhaps to the failure in preparing the injection material correctly.

4. Injection with starch red lead and barium sulphate and then X-raying proved satisfactory for demonstrating the vessels in skeletal muscle.

The experimental work was divided into four sections:

1. The placental vessels
2. The pulmonary bronchial vessels
3. The intestinal vessels
4. The vessels of muscles.

In each section a preliminary discussion on the results of previous workers preceded the records of the experiments performed, and was concluded by review of the facts observed. 23 photographs and 59 drawings illustrated the results obtained.

From a review of the experiments performed, the conclusion was drawn that there is a very close relationship between the course of the vessels and the function of the organs they supply. The observation that the course of arteries is always protected as far as possible, was repeatedly demonstrated in these experiments. Especially was this so in relation to the arteries supplying muscles. Muscles of similar form have a similar pattern of blood vessels. The vessels which go to a muscle do not traverse a contractile element (another muscle) first which would act as a living ligature, unless the muscle they traverse is part of the muscle they are going to supply. No vessels were seen traversing muscles of a different structure and function to the one they were going to supply.

The relation of the course of arteries to the movements of the organs they supply was demonstrated. The anastomotic channel between the/

the two umbilical arteries as they enter the placenta, the constant anastomotic arcade across the bifurcation of the main arteries of the colon, the anastomotic loops between the nutrient vessels entering the quadrilateral and fusiform muscles are all designed with the similar purpose of regulating the flow through them as the wave of contraction passes along the region they supply. The arterial patterns are, in fact, intimately related with the movements of the organs they supply. In the more fixed parts of the colon there is a straight marginal artery with the vasa recta coming off it. When an area is reached where there is more mobility, i.e. the pelvic colon, arcade formation starts in the pelvic mesocolon, similar, though not as complicated as in the mesentery of the small intestine, also a mobile organ. The vessels on the intestine walls are not mainly longitudinal channels in which a concertina like effect would be produced on them when waves of peristalsis pass down the intestine but are in line with the circular muscle of the intestine, as a series of vascular rings. This arrangement also suits the venous return, the veins having the same course as the arteries.

The blood supply to sartorius is also comparable with this - not one long vessel arising at/

at the top and running down in the contractile element of the muscle from origin to insertion but a series of vessels entering all the way down with the finer subdivisions running parallel with the muscle fibres.

Anastomoses are not haphazard affairs. The constant presence of an anastomic channel across the bifurcation of an artery, in the vessels of the intestine, was noted in all the specimens examined. No specific search was made for Sudeck's "critical point", a point on the pelvic colon where the vascular supply is stated to be especially precarious on account of the poor anastomosis between the superior haemorrhoidal and the lowest sigmoid artery. But in the specimens of colon examined (Nos. 4, 9, 13, 14, 15a, 16, 17, 24) which included that area of the intestine, the arcade produced by an anastomotic channel across the bifurcation between superior haemorrhoidal and last sigmoid artery was similar to those between the other sigmoid arteries. There is no reason why there should not be this anastomosis - the absence of an anastomosis would be contrary to the usual formation, and certainly in these experiments Sudeck's point was not present.

An/

An anastomotic channel between the peripheral ends of the umbilical arteries would be in a precarious position, constantly occluded by the waves of contraction passing over the uterus, and also hindering the regular and orderly thrombosis in the vessels of the cotyledons when the placenta separates, which are specially designed as end arteries to produce this effect. No such channel was present in any of the specimens injected in the present investigation. Similarly there is no anastomosis of arterial size between the pulmonary and bronchial arteries. These vessels have very different functions, and a free anastomosis between them is therefore not normally required. No such anastomosis was demonstrated.

Some facts, as described by previous investigators were confirmed, while with others different results were obtained. In brief these may be summarised as follows:-

1. The findings regarding the placental vessels were the same as those of Bacsich and Smout, as I have described in the section on the placenta, viz. the presence of a communicating channel between the two umbilical arteries, as they enter the placenta, and no anastomoses thereafter.

2. The absence of arterial anastomosis between the pulmonary and bronchial arteries, was confirmed. This result concurs with the work of Daly and W.S. Miller.

3. A free anastomosis was found between the vasa recta of the small intestine, on the intestinal wall and between vasa recta of opposite sides on the antimesenteric border. This confirms the findings of Noer, and is different to the older observations of Cokkinis, who found no such anastomoses.

4. The loop formation of the vasa recta on the base of appendices epiploicae, as described by Meilleire, was not observed in any of the 26 colons examined. Experiments were performed in tying the base of several appendices epiploicae prior to injection, to see if these affected the vasa longa; but in every case the injection material flowed on beyond the level of the ligature.

5. A different approach to other investigations was taken in the study of the vascular supply to skeletal muscle. Some muscles were taken according to their classification by the arrangement of their component fibres and the arterial pattern in individual muscles from different parts of the body belonging to the same group were compared.

The arterial supply of muscles was classed as (1) major (2) minor supply. Four varieties of arterial pattern were distinguished, not five as Blomfield has described them. It was considered that the distinction of the anastomosis between entering vessels in certain muscles, either as a longitudinal channel or as a series of loops, into two separate groups was an unnecessary elaboration.

The/

The two specimens of adult limbs had been amputated for conditions which were not considered to have affected the vascular patterns, and the results obtained were considered to demonstrate a more or less normal appearance, and could be used to compare with those of a limb removed for vascular disease. The present investigation was an enquiry into normal appearances. Hence several limbs amputated for senile gangrene, though available were not utilised as the appearances they would have shown could not have demonstrated what was desired, the normal pattern.

A number of variations were noted especially in the origins of the vasa recta of the colon, some arising singly, some arising as a pair off a common stem and going to the same or to opposite sides of the intestine. But these are minor variations, and the essential underlying pattern - straight vessels passing at right angles to the long axis of the colon with any given area of colon wall getting approximately the same supply as another similar area was the same, whatever the origin of the feeding arteries may have been. The same applies to the origin of vessels off an "axis" artery in the limbs - the superficial epigastric, circumflex/

circumflex and external pudendal arteries coming off the profunda femoris for example, as noted on one cadaver recently, is merely a variant and is perfectly normal. Such alternate patterns to those more usually described should be remembered, however, and students weaned of the idea that they are "abnormal". In preference the term "variation" should be employed.

One must accept the "dynamic view" of the vascular system, as Woollard (1922) has described it "The vascular net depends upon the inherent properties of certain cells to form blood vessels and blood cells, these properties being regulated by the needs and activities of the surrounding tissues". There is nothing haphazard or irregular about it. "Function determines form"; and in the end arteries of the placenta, the rich vascularity of the small intestine with its freely communicating arterial rings, and the other examples described in the previous pages, this law is displayed.

§ § § § § § § § § §

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APPENDIX I.

P 54. Placenta (1). Fresh specimen 16.2.46.

Cannulae tied into both umbilical arteries. Perfused through with tap water till umbilical vein outflow ran clear. Then injected with 6% celloidin in acetone stained with alkanin at a pressure of 300 mm HG for half an hour. Pressure left on at 300 mmg for 48 hours. Specimen then placed in 50% HCl on 18.2.46. Washed with water on 26.2.46 and replaced in acid. 28.2.46 washed again till clean.

P 54. Placenta (2). Fresh specimen 18.2.46.

Cannulae tied into both umbilical arteries. Perfused through with tap water till umbilical vein outflow ran clear. Then injected 18.2.46, one umbilical artery with 6% celloidin stained with alkanin, the other with 6% celloidin stained with trypan blue. Injection with red started first, and this began to flow back along the second artery before the blue injection tube was connected. Pressure was left on at 300 mm Hg for half an hour.

There was some leakage in the tubing. Pressure was therefore not maintained and the main vessels did not fill out sufficiently to form a firm cast. Placed in 50% HCl 20.2.46 after 24 hours in water. Washed with water 11.3.46.

Pp 55 and 56. Placenta (3). Fresh specimen 20.2.46.

The right and left umbilical arteries were traced down in the umbilical cord to within 1" of the placenta, where the communicating artery was found. This was ligated and thus the two umbilical arteries were separated. Cannulae were inserted and the vessels were perfused for two hours with acidulated water. Acetone was then injected to enable the celloidin acetone mixture to flow freely to the terminal ramifications before coming out of solution.

One umbilical artery was injected for half an hour at 300 mm Hg with 6% celloidin acetone, stained with trypan blue. Pressure was left on at 250 mm Hg. On 21.2.46 after 12 hours 20 mm Hg pressure was still present, but there was no firm coagulum in the main vessels yet.

The/

The second artery was then injected with 6% celloidin stained with alkanin, quarter of an hour at 300 mm Hg, and one hour at 200 mm Hg. After 3 hours the pressure was raised to 300 mm Hg, and left.

On 22.2.46 a firm cast had been obtained of the alkanin-stained artery. The blue artery's main trunks were still not solid; it was connected up again, and further trypan blue celloidin (12%) injected at 300 mm Hg for fifteen minutes, then pressure left on at 200 mm Hg. After four hours the pressure still read 150 mm Hg, and after 24 hours it stood at 60 mm Hg.

23.2.46. The blue artery was now solid. Pressure was left on till 25.2.46 when the specimen was placed in 60% HCl.

On 6.3.46 the specimen was washed in water, and replaced in acid.

11.3.46. Rewashed with water. A satisfactory corrosion cast was obtained.

P 57. Placenta (5). Fresh specimen 22.4.46.

Washed through with acidulated water.

Injected on 23.4.46 with a mixture of 6% celloidin and 3% camphor in acetone, stained with alkanin. Pressure at 400 mm Hg for half an hour, then left on for 24 hours.

26.4.46. Placed in 75% HCl.

29.4.46. Washed with water. "Durofix" used to strengthen the main vessels, where the cast was not quite solid.

APPENDIX II.

P 67. Lung Experiments

(a) Pulmonary artery trunk in child aet 3, killed in accident.

Injected at pressure of 200 mm Hg for one hour with 6% celloidin, stained alkanin, after preliminary perfusion with water. No pressure left on. Unsatisfactory result.

(b) Specimen "pluck" of lungs, trachea, oesophagus and aorta.

Vessels of aortic arch tied, and intercostals ligated 1/3" out from aorta.

Aorta injected with 6% celloidin stained alkanin at a pressure of 200 mm Hg for half an hour. Leakage occurred. Unsatisfactory result.

(c) Still-born infant. Similar experiment as (a) and (b), also unsatisfactory.

(1) Child aet 3, "pluck" of lungs, trachea, oesophagus and aorta. Aorta injected as in (a). Bronchial arteries failed to inject.

(2) Still-born infant. Similar experiment as (1). The ductus arteriosus was not ligated, and a mixed injection occurred.

(3) (4) and (5) Pluck of both lungs, trachea, oesophagus, and thoracic aorta from still-born infants, perfused with water, then injected with 6% celloidin stained alkanin for 1 hour at 200 mm Hg pressure, pressure left on for 12 hours, then placed in acid 50% HCl for a week. Unsatisfactory as the specimens broke up following corrosion.

(6) Similar specimen as (2), (3), (4) and (5), treated same way. A good cast was obtained of both pulmonary arteries.

(7) Similar experiments, specimen leaked. It was therefore dissected without corrosion.

P 67. Specimen (7). The Bronchial Arteries

The right lung.

The upper right bronchial artery sprung from the second right aortic intercostal and ran over the oesophagus and trachea and vagus, supplying all three, and then on to the upper bronchus. The lower bronchus was supplied by an artery coming from the other side.

P 68. Specimen (8).

Aorta injected to show the bronchial arteries, with 6% celloidin, stained alkanin, as in (3), (4) and (5). Specimen leaked as the innominate artery had not been tied off tightly enough. Was dissected therefore without maceration in acid.

P 68. Specimen (9).

The pulmonary artery injected with 6% celloidin, stained alkanin, pressure 200 mm Hg one hour. Pressure left on 16 hours then placed in 50% HCl for one week.

P 68. Specimen (10).

Pulmonary trunk and aorta injected separately after ligating the ductus arteriosus, same technique as in (9). Old X-ray films were used to supply the celloidin.

P 68. Specimen (11).

Pulmonary trunk injected. Same technique as (9) using pure celloidin.

P 68. Specimen (12).

Both pulmonary and bronchial systems injected as in (10). Some leakage occurred and the specimen was a failure.

P 69. Specimen (13).

Pulmonary and bronchial arterial systems injected as in (10), using pure celloidin, not X-ray plates celloidin.

P 69. Specimen (15).

Cannulae into right and left pulmonary arteries and aorta. Injected for half an hour at 400 mm Hg, five minutes at 500 mm Hg. Pressure left on 17 hours. No leak.

P 69. Specimens (16) and (17).

Same technique as (15). Considerable leakage occurred in both these specimens, due to trauma of the aorta.

In No.(16) the pressure dropped 100 mm Hg in two hours.

P 70. Specimen (20).

The aorta was injected with 6% celloidin stained with alkanin, the pulmonary artery with 6% celloidin stained with trypan blue.

APPENDIX III.

Colon Experiments

P 86. Colon (1).

Injected for 20 minutes with a 15% solution of celloidin in acetone at a pressure of 200 mm Hg.

Pp 86 and 87. Colon (2).

The ileocolic artery; middle colic and first sigmoid arteries were each injected for half an hour with a 15% solution of celloidin in acetone, stained with Sudan III, at 170 mm Hg pressure, the pressure being left on in the middle colic artery for 15 hours.

P 88. Colon (3).

Loop of colon filled with water, and floated in water during injection. Injected twelve hours after death for half an hour at 400 mm Hg with 6% celloidin, pressure left on 14 hours. Right colic artery injected at its bifurcation into upper and lower divisions. Mid colic artery's trunk ligated half an inch from bifurcation, and a second "mid" colic artery ligated four inches from main mid colic artery, also ligated a quarter of an inch from its bifurcation. "Marginal" artery ligated at both ends.

P 89. Colon (4).

Injected 24 hours after death at a pressure of 500 mm Hg for twenty minutes and pressure left on fifteen hours. Gross leakage occurred.

P 90. Colon (5).

First sigmoid artery injected 32 hours after death with celloidin 5%, pressure left on for 24 hours.

P 91. Colon (6).

Injected 24 hours after death with 5% celloidin in acetone, stained with Sudan III for half an hour at a pressure of 400 mm Hg., and pressure left on for 12 hours.

P 92. Colon (7).

Left colic artery injected 24 hours after death with 5% celloidin in acetone, stained Sudan III, for half an hour at 400 mm Hg pressure and pressure left on for 12 hours.

P 93. Colon (8).

Injected with 5% celloidin for half an hour at 400 mm Hg and pressure left on for 14 hours.

P 94. Colon (9).

Inferior mesenteric artery injected 12 hours after death with 5% celloidin stained with Sudan III for half an hour at 400 mm Hg, pressure left on 12 hours.

P 95. Colon (10).

First sigmoid artery injected with 6% celloidin stained with Sudan III for half an hour at 400 mm Hg, pressure left on 12 hours. Macerated in 50% HCl for 16 hours.

P 95. Colon (11) Male aged 65. Died of carcinoma of lung.

Specimen - mid transverse colon.

Mid colic artery injected with 6% celloidin stained with Sudan III for half an hour at 450 mm Hg. Pressure left on for 12 hours. Corroded in 50% HCl for three days. Numerous adhesions in the omentum attached to this specimen, but no vascularisation was visible. Further notes lost.

P 95. Colon (12)

Descending colon injected with 6% celloidin stained with Sudan III for half an hour at 400 mm Hg. Pressure left on for 12 hours. Left 6 hrs. in cold water then corroded one week in 50% HCl 6 hours in 75% HCl.

P 96. Colon (13)

Inferior mesenteric artery injected with 6% celloidin stained with alkanin for half an hour at 200 mm Hg, pressure left on for 16 hours then placed in 75% acid.

P 97. Colon (14)

Inferior mesentery artery injected with 6% celloidin stained with alkanin for half an hour at 200 mm Hg pressure. Gross leakage.

P 98. Colon (15)

Injected with 6% celloidin in acetone, stained alkanin half an hour at 400 mm Hg, pressure left on 12 hours. Placed in 50% HCl for one week.

P 99. Colon (15a)

Aorta injected with 6% celloidin stained with alkanin for half an hour at 500 mm Hg and pressure left on 12 hours. Then placed in 50% HCl for 48 hours.

P 100. Colon (16)

Inferior mesenteric artery injected with 6% celloidin in acetone stained with alkanin for half an hour at 500 mm Hg pressure, and pressure left on for 12 hours.

P 103. Colon (20)

Full term male foetus.

Injected well. In acid usual technique.

P 104. Colon (21)

Injected 12 hours after death. Specimen injected with 6% celloidin stained with alkanin for half an hour at 400 mm Hg, pressure then left on 12 hours. Specimen placed in 50% HCl one week, then washed in gentle stream of running tap water.

P 105. Colon (23)

Injected 18 hours after death with 6% celloidin stained with alkanin for half an hour at 400 mm Hg pressure, five minutes at 500 mm Hg, and pressure left on. In 50% HCl for 9 days then washed with H_2O.

P 106. Colon (24)

Aorta injected with 5% celloidin stained with alkanin for half an hour at 400 mm Hg. Pressure left on 12 hours.

P 107. Colon (25)

Left colic artery injected with 6% celloidin stained with alkanin for half an hour at 400 mm Hg pressure. Pressure left on 12 hours. Partially corroded in 50% HCl.

P 108. Colon (26)

18.12.45 Perfused with water.

19.12.45. Injected with 6% celloidin, stained with alkanin for one hour at 250 mm Hg. Pressure left on at 200 mm Hg. After 18 hours still 30 mm Hg. Placed in 50% HCl. 27 days later removed from acid and washed with water.

P 109. Small Intestine No. (1).

Injected 12 hours after death on 8.8.39. Jejunal artery injected with 6% celloidin stained with alkanin for half an hour at a pressure of 400, pressure left on for 12 hours. Specimen then placed in 50% HCl for 14 days.

P 110. Small Intestine No.(2).

18.12.45. Perfused with water. Jejunal artery injected with 6% celloidin stained with alkanin for one hour at 200 mm Hg. Pressure left on 18 hours. Some leakage occurred around the vessels, due to post mortem changes, the specimen being a week in H_2O before injection. Placed in 50% HCl. Removed after 19 days.

P 111. Small Intestine No.(3)

Perfused with water 24 hours after death. 18.12.45. Jejunal vessel injected with 6% celloidin stained with alkanin for one hour at 250 mm Hg, pressure being left on at 200 mm Hg. Pressure read at 30 mm Hg after 18 hours. Placed in 50% HCl for 41 days.

P 112. Small Intestine (Canine specimen).

Aorta injected 9.3.46 with 6% celloidin stained with alkanin at 400 mm Hg for half an hour, pressure left on 12 hours.

APPENDIX IV.

MUSCLE VESSELS EXPERIMENTS

P 129. Foetus (A).

Perfused with water till return flow from inferior vena cava ran clear, then injected with 6% celloidin in acetone stained with alkanin for half an hour at 200 mm Hg pressure. Pressure left on for 4 hours.

Left lower limb then removed and placed in acid, 50% HCl, on 12.12.45; washed with water, and the superficial layers of skin and muscle thus removed 17 days later. Rinsed with water again 17 days later, i.e. after 34 days corrosion.

P 130. Experiment (3).

Injected 24 hours later on 14.12.45. Vessels perfused with water and then injected with 6% celloidin stained alkanin for half an hour at 200 mm Hg. Pressure left on for 4 hours at end of which time it had fallen to nil. Placed in 50% HCl for 15 days and then, as it was still practically unaffected by this concentration, placed in 100% HCl for 48 hours. This produced rapid charring and corrosion of the specimen.

P 131. Experiment (4).

Popliteal artery perfused with water 24 hours after death. Then injected with 6% celloidin stained alkanin for one hour at 250 mm Hg, and pressure left on at 200 mm Hg for 48 hours. In water for 4 days then placed in 50% HCl for 26 days. Rinsed with water, and corrosion cast obtained.

P 132. Experiment (5).

Aorta perfused 3.1.46 with water, till return from inferior vena cava ran clear. Right external iliac artery then injected with 6% celloidin stained alkanin for half an hour at 200 mm Hg, and pressure left on for 17 hours. Placed in 50% HCl for 28 days.

P 134. Experiment (7).

Aorta perfused with water on 3.1.46 till return from inferior vena cava ran clear.

Right external iliac artery then injected with 6% celloidin stained alkanin for half an hour at 200 mm Hg pressure, pressure left on for 17 hours Placed in 50% HCl for 28 days.

P 138. Experiment (11).

On 29.3.46 left leg femoral artery was injected with 6% celloidin stained alkanin for half an hour at 400 mm Hg. Pressure left on 48 hours. Then placed in 75% HCl. Washed in water after three days, and re-inserted in acid for further two days.

P 141. Experiment (14).

Popliteal artery perfused with acidulated water three days after amputation till the venous return was fairly clear. Then injected with starch red lead barium sulphate mixture (4:1) for quarter of an hour at 400 mm Hg pressure and pressure left on. 12 hours later the pressure still stood at 380 mm Hg. The injection was stopped.

P 153. Experiment (26).

29.3.46. Left subclavian artery was injected with 6% celloidin stained alkanin for half an hour at 400 mm Hg pressure. Pressure left on for 48 hours, then placed in 75% acid. Washed in water after three days and replaced in acid for a further three days. Washed in water.

I N D E X.

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